THE PRINCIPLES OF
ANTE-NATAL & POST-NATAL CHILD HYGIENE

BY

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With 161 illustrations and 14 plates, including over 100 portraits

"Are you not risking the greatest of your possessions? For Children are your riches, and upon their training for well or ill depends the whole order of their father's house."

—SOCRATES' APPEAL TO THE SENATE

PHILADELPHIA
F. A. DAVIS COMPANY, Publishers

1929

MADE AND PRINTED IN GREAT BRITAIN BY
JOHN BALE, SONS AND DANIELSSON, LTD.,
LONDON, W.1.

TO

Hilda

IN

CELEBRATION

OF THE

24TH AND 25TH OF SEPTEMBER

PREFACE

THIS book is meant to be a companion volume to my book on "The Principles of Ante-natal and Post-natal Child Physiology," with which it is uniform in size and manner of treatment, and, like it, is, I believe, the first of its kind in any language. It is comprehensive and is designed to appeal to every educated and thoughtful person, lay or professional, who is more than superficially interested in the welfare of children during their various phases of ante-natal and post-natal life. In addition, it is hoped that pædiatric physicians, medical officers of ante-natal and of infant and child welfare centres, as well as students engaged in original investigations in connection with the hygiene of child life, will find this volume a serviceable book of reference. The latter, indeed, should find it a very useful guide, inasmuch as it summarises critically and in considerable detail practically everything that has been done in the various branches of Child Hygiene up to the date of publication. Lastly, I believe that the book will be found indispensable to candidates reading for the projected special diploma in Ante-Natal Care, Midwifery, and Infant Welfare.

The chapter on "The History of Child Hygiene," as well as the historical matter interspersed here and there throughout the book, will be found of interest by all intellectual readers, whilst the chapter on "Biometrics" will, I trust, be appreciated by all who are willing to weigh statistical evidence judicially before accepting it as decisive.

The references to literature have been given in accordance with the following scheme: Papers which are interesting, but not of very great importance from the point of view of the subject under consideration, are referred to in the body of the book under the names of their authors and the dates of their publication, e.g., Heape in 1890, Castle and Phillips in 1911, &c. These details are sufficient to enable one who is sufficiently interested, and in touch with a good library, to locate the original works without much difficulty. Papers which I was of opinion were

necessary to extend the reader's horizon with respect to any point under discussion, I have given as numbered references at the bottom of the page. In addition, I have appended references to books and monographs, fairly easily accessible, at the end of each chapter. These will furnish the reader not only with fuller information on the subject of the chapter, but will also provide him with a full bibliography on the subjects discussed in that chapter. By adopting this plan I have, I hope, succeeded in giving the reader a complete bibliography without overburdening the book with a bewildering number of references which might prevent him from seeing the wood for the trees.

A word of explanation is perhaps needed with regard to the portraits reproduced in this volume. In the case of dead pioneers, either recent or remote, I have reproduced their portraits whenever I could get hold of them. For these I am indebted to the Council of the Royal Society of Medicine for putting their splendid collection at my disposal. Unfortunately there are, as far as I know, no portraits obtainable of some of the most eminent of the ancient and mediæval pioneers, such as Soranus, Thomas Phaer, Michael Underwood, &c. With regard to living authorities, whilst all whose portraits are included have done important work in connection with preventive pædiatrics, the reader will perhaps wonder why the photographs of some English authorities, whose names are constantly on the lips of every student of child medicine, have been omitted. The explanation is twofold. In the first instance, I have not included the portraits of any pædiatrician, however famous, whose claim to celebrity lies in the domain of purely clinical pædiatrics, but who has contributed little to our knowledge of child nutrition or child hygiene. Secondly, paying regard to the strict ethical code governing the medical profession in England, I have limited myself to those authorities who for one reason or another are not engaged in private medical practice, and I herewith take the opportunity of thanking many of these English as well as foreign authorities for their courtesy in sending me their photographs for this book. I also wish to thank the following friends for procuring for me a number of portraits: Professor W. Bulloch, Dr. Margaret Lowenfeld, Dr. J. D. Rolleston, Dr. C. W. Saleeby, Dr. M. Marcus, Dr. L. Spira, Dr. A. Levinson, of Chicago, and Dr. Gustav Dinolt, as well as Herr Max Schneider (photographer), of Vienna. The publishers, Messrs. John Bale, Sons and Danielsson, have also kindly procured for me a certain number of portraits of living

authorities. Messrs. W. B. Saunders and Co., Herr Julius Springer, and Messrs. Longmans, Green and Co., have kindly allowed me to reproduce a number of portraits from Garrison's chapter on "History of Pediatrics," in vol. i of Abt's "Pediatrics," Bokay's "Die Geschichte der Kinderheilkunde," and my own "The Principles of Ante-natal and Post-natal Child Physiology" respectively. I further owe a debt of gratitude to Dr. Ulysses Williams for the beautiful skiagram of Plate XIV, to the Authorities of the British Museum for the facilities they gave me for reproducing the Roman coin showing Puellæ Faustininæ (fig. 16, p. 42) and to the Editor of *Maternity and Child Welfare* for the use of a number of illustrations. The sources of other borrowed illustrations are acknowledged in the body of the book. Lastly, I wish to thank Professor F. J. Browne for having kindly read the proofs of the ante-natal portion of the book, and Dr. Harry Fisher, D.M.R.E., Dr. Jack Freudenheim, and my son Vivian, for some help they gave me whilst the book was passing through the press.

W. M. FELDMAN

*Harley Street, W.*1.
September, 1927.

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ERRATA

Page 40, in legend to Fig. 14A—H. Pless *should be* H. Ploss.
,, 44, FIG. 8 *should be* FIG. 18.
,, 54, line 8 from bottom—Babinsky *should be* Baginsky.
,, 63, first reference to literature—Pignut *should be* Pignat.
,, 66, line 8, "individual" *should be* "all."
,, 73, in column (3) of table—(o) *should be* (h).
,, 74, 10th line from bottom—σ_{om} *should be* r_{om}.
,, 76, in example (3) — y = 68·65 =, &c., *should be* y − 68·65 =, &c., *throughout*, *thus*: $y - 68{\cdot}65 = 1{\cdot}20$, $\therefore y = 69{\cdot}85$.
,, 86, line 10—"see fig. 22" *should be* "see fig. 23."
,, 120, paragraph on umbilical knots—With two slack knots a superimposed pressure, &c., *should be* With two slack knots superimposed, a pressure, &c.
,, 128, line 7 from bottom—*add the words*, "together with those of Dr. Meyer for New York City," *after* XXXIII, &c.
,, 180, last line—p. 170 *should be* p. 140.
,, 182, the whole of the first sentence of the last paragraph *should be omitted*, and the words, "as seen from Table LXXIX" *should be inserted in the last line after the words* "England and Wales."
,, 192, first reference to literature—Ashby, Henry, *should be* Ashby, Hugh Tuke.
,, 200, 16th line from top should begin "*is* 0·216 ± 0·058."
,, 222, Table LXXXVII *should be* Table LXXXVIII.
,, 242, 1st word in last line *should be* "in."
,, 257, 9th line from bottom—"that" *at the beginning of the line should be omitted.*
,, 258, line 4 from top—Slemms *should be* Slemons.
,, 268, line 7 from top—Allen, Thomson, *should be* Allen Thomson, *and* Bovers *should be* Boveri.
,, 268, last reference to literature — Hemons *should be* Slemons.

Page 293, reference 1—*Zeitr. should be Beitr.*

,, 335, line 7 from bottom — Vitamins A and C *should be* Vitamins A, C and D.

,, 394, legend to Fig. 98—Jaschne's *should be* Jaschke's.

,, 403, in Table CXXXI, percentage of caseinogen in human milk *should be* 1, *not* 7.

,, 413, footnote, p. 88—*should be* p. 881.

,, 433, line 18 from top — Joseph Bannermann *should be* Joseph Brennemann.

,, 446, under literature—Discussion on the Modification of Cow's Milk *should be a separate entry at the end. It has no relation to* D. Paterson.

,, 465, under literature—"Mothercraft" Lectures, &c., *should be a separate entry. It has no relation to* M. Lidiard.

,, 682, last line in paragraph on Epilepsy—the comma after strong *should be omitted.*

PART I

PROLEGOMENA

"Parents should be cleanly wed,
Children must be nobly bred,
Wisely fed and firmly led."

JOHN BURNS.

THE PRINCIPLES OF

ANTE-NATAL AND POST-NATAL CHILD HYGIENE

CHAPTER I

INTRODUCTORY AND GENERAL SURVEY

"The bridge thou seest, said he [the genius], is human life; consider it attentively. Upon a more leisurely survey of it I found that it consisted of three score and ten entire arches, with several broken arches which added to those that were entire made up the number about an hundred. . . . . But tell me further, said he, what thou discoverest on it. I see multitudes of people passing over it said I, and a black cloud hanging on each end of it. As I looked more attentively, I saw several of the passengers dropping through the bridge, into the great tide that flowed underneath it; and upon further examination, perceived that there were innumerable trap-doors that lay concealed in the bridge, which the passengers no sooner trod upon but they fell through them into the tide and immediately disappeared. These hidden pitfalls were set very thick at the entrance of the bridge, so that throngs of people no sooner broke through the cloud, but many of them fell into them. They grew thinner towards the middle but multiplied and lay closer together towards the end of the arches that were entire."

JOSEPH ADDISON, "The Vision of Mirzah."

Child Hygiene concerns itself with everything that tends to preserve the life, health and welfare of the child during its various stages of ante-natal and post-natal development. When towards the end of the last century the subject of child welfare began to engage the serious attention of physicians, politicians, and sociologists in different parts of the world, it was thought that the earliest time at which it was necessary to begin to deal satisfactorily with the welfare of the child was the moment of its birth. It was soon realised, however, that birth is not "the point at which we begin to live," and that to make a proper beginning we must start not from the moment of birth which is only one of the turning points in the life cycle of the child, but from the moment of conception—when by the fusion of the minute sexual

cells, the ovum and spermatozoon, coming from the mother and father respectively, the child truly starts its career as a new being.

It is the period in the child's life cycle, comprised between conception and birth—i.e., the intra-uterine stage of development — which is the most important period in the child's career. It is during that time that the child goes through by far the greatest part of its development. For whilst from the moment of birth till adult age the child increases about twenty-fold in weight (i.e., from $3\frac{1}{2}$ kilograms to 70 kilograms), and no more than about three and a quarter times in length (i.e., from 50 cm. to about 165 cm.), the increase in weight from conception to birth is no less than 900 million-fold and its increase in length is approximately 2,500-fold! The human ovum, at the moment it is fertilised by the spermatozoon and is ready to begin its series of wonderful kaleidoscopic transformations which ultimately result in the production of a complete human infant, is a small spherical vesicle measuring no more than $\frac{1}{3}$ mm. in diameter, and of almost imponderable mass—its weight being about $\frac{1}{250}$ mg. This exceedingly minute cell is converted from an inert and vegetative being into something extraordinarily active by the energising agency of the head of the spermatozoon whose mass is only about $\frac{1}{200000}$ part of that of the ovum. This insignificant sperm cell imparts an impetus to the inert ovum, changing its potential into kinetic energy and setting it upon its career of almost incessant activity, which causes it to be converted from a simple unicellular organism into a complicated multicellular human infant of a weight hundreds of million times that of the ovum from which it sprang. As Dr. Charles Mercier picturesquely puts it: "The female element is the coals in the grate; the male element is the match that sets them alight. The female element is the great mass of dough in the kneading trough; the male element is the little bit of yeast that leavens the whole lump and sets it all fermenting."[1]

We see, therefore, that considered from the point of view of increase in bulk alone, the intra-uterine stage is a period of tremendous activity in the child's career, and that to begin to supervise the child after it has been born is to begin at a period when it has already gone through by far the greatest part of its growth and development. Moreover, whilst during its

[1] Charles Mercier, "Heredity and Inheritance as they Concern the Physician," *Lancet*, 1913, ii, p. 1300.

post-natal and later intra-uterine life, the child's development simply consists of growth along fixed and definite lines, the period of development immediately following conception is characterised not only by the conversion of a single cell into a vast number of regularly arranged cells, but also by the grouping and disposition of these cells into masses which go to form organs. This particular epoch of which we are speaking lasts six weeks, and has been subdivided by Ballantyne into two periods called respectively the *germinal* and the *embryonic* periods, and is of particular importance in the subject of child hygiene, since it is during the embryonic period that congenital malformations are produced.

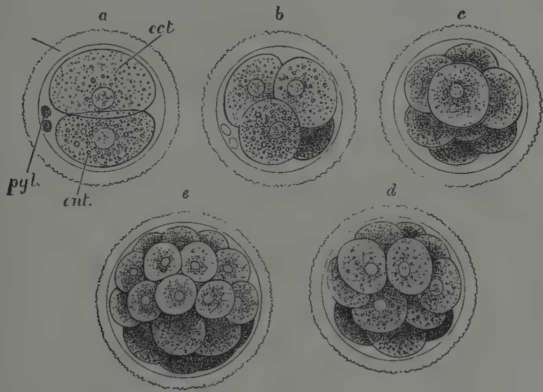


FIG. 1.—Segmentation of ovum (Quain's "Anatomy").

The Three Periods of Intra-uterine Life.

(*a*) **Germinal Period.**—This is the period, occupying about one week, occurring immediately after fertilisation. During this stage, the single-celled ovum begins to segment. It first divides into two cells, the two new cells again divide, giving rise to four cells, and so on in geometrical progression until it is converted into a solid mass or globe called the morula—or little mulberry (see fig. 1).

The inner cells of this solid mass, being deprived of nourishment derived from the maternal blood which bathes the ovum, soon atrophy and die and their place is taken by fluid. The morula therefore becomes transformed into a hollow vesicle or blastula (fig. 2). At a certain area on the surface of the vesicle

a number of cells become particularly active and it is from these that the future human being develops. This area is called the *embryonic area* (fig. 3).

The germinal stage, then, of intra-uterine life consists of the period between fertilisation and the appearance of the embryo in the embryonic area of the blastula.

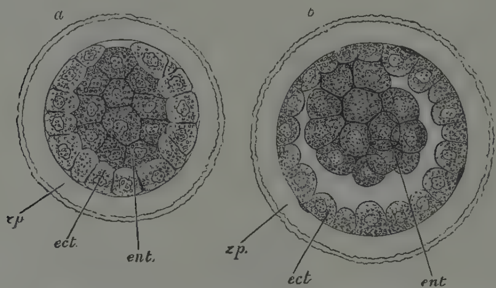


Fig. 2.—Section of the ovum of the rabbit during the later stages of segmentation, showing blastula stage (Quain).

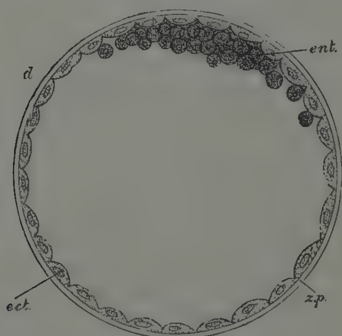


Fig. 3.—Bilaminar blastoderm; *ect* = ectoderm, *ent* = endoderm (Quain's "Anatomy").

(*b*) **Embryonic Period.**—This period which follows immediately upon the last and extends up to about the sixth week of pregnancy, is characterised by the arrangement of the various cells into groups to form organs (see fig. 4). It may, therefore, be called the organogenetic period or period of differentiation, and it owes its great importance, from the point of view of child

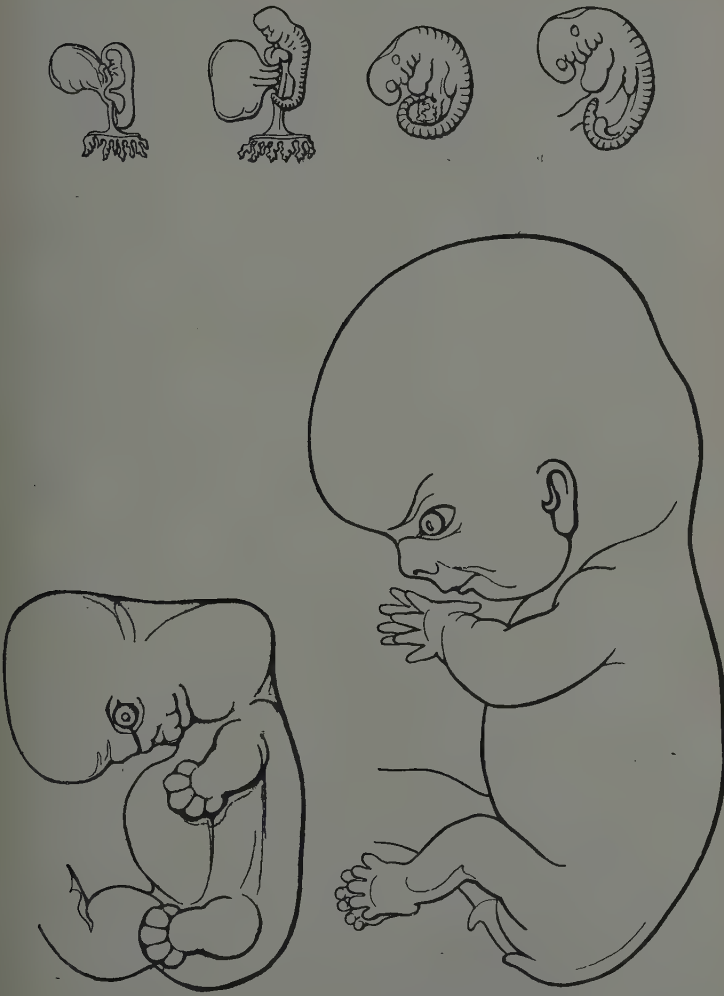


FIG. 4.—Different stages of embryonic development from about the fourth to the eighth week (four times natural size).

hygiene, to the fact that any derangement in the disposition of the cells during this stage, arising from any cause, results in the production of monstrosities or congenital malformations. The nature of all the disturbances in the normal organogenesis of an individual before birth which are responsible for *teratogenesis*, i.e., the production of monstrosities (from the Greek τερας, a monster, and γεννάω, I beget) is not yet understood. Some, as we shall see, are of an inheritable nature, being transmitted from parent to offspring in accordance with the laws of heredity; but others quite certainly result from the action of poisons or toxins circulating in the maternal blood from which the embryo derives its nourishment. Experiments have shown that not only can monstrosities be produced in birds by varnishing over part of the shell of an incubating egg so as partially to deprive it of oxygen, or by injecting poisons, toxins, microbes and various alcohols into the egg, and in aquatic animals by altering the chemical composition of the fluid in which the eggs develop, but that by the administration of excess of alcohol to pregnant mammals in the early stages of pregnancy various malformations can be produced in their developing embryos (see pp. 111-113). Similar results can be produced in avian eggs by raising the temperature of incubation to a degree above the normal incubation temperature, but not high enough to kill the egg. From this it follows that if the blood of an expectant mother in the early stages of pregnancy is deficient in oxygen—e.g., in cases of severe heart disease—or contains some toxin such as that of syphilis, tubercle, lead or alcoholic poison, &c., or if it is of a continually higher temperature than normal, such as is the case in various pyrexial diseases, her developing embryo will suffer. If the concentration of the toxin or poison in the maternal blood is sufficiently strong, or if the temperature is sufficiently high, development may be completely arrested and the embryo will be killed outright; if, however, the concentration of the toxin is more dilute, or the blood temperature is not much higher than normal, the arrest in the embryonic development is partial only so that merely a portion of the body becomes delayed in its development, with the result that a malformed infant is produced. Indeed, in the case of human beings it is not uncommon to find some association between fœtal malformation and some toxic disease in the mother, such as syphilis, tubercle, lead poisoning, alcoholism or infectious disease either before or during pregnancy. If we could obtain a continuous

cinematographic record of the embryological or developmental processes of the unborn infant, "we could recognise when and in what part of the living picture the process of arrestment took place."[1]

(c) **Fœtal Period.**—The period between the embryonic stage and the end of pregnancy is called the *fœtal period.* During this stage there is no further differentiation of cells to form new organs, since the formation of organs has already been completed in the embryonic stage. It is important to remember this fact from the point of view of *maternal impressions*, which we shall discuss more fully in another chapter (see Chapter IX). Since all the main parts of development or organogenesis have been completed by the end of the second month of pregnancy (see fig. 4) it is clear that no malformations (as opposed to deformity due to mechanical or pathological causes) can arise from impressions received after the second month. The chief function of the organism during the fœtal period is growth along the lines laid down in the previous organogenetic stage. In the early part of this period an intimate connection is established between the maternal and fœtal bloods through the placenta or after-birth which acts as the composite organ of nutrition, respiration and excretion of the fœtus. Deformities may, and do, occur during this stage of the child's development, but as far as we know such deformities are produced by:—

(a) *Mechanical Causes.*—These may operate either intra-abdominally, e.g., abdominal tumours, narrowness or malformation of the uterus, oligo-amnios (i.e., deficiency of liquor amnii), extra-uterine pregnancy, coiling of the umbilical cord round a limb, causing spontaneous amputation, &c.; or the pressure may be exerted upon the fœtus extra-abdominally, e.g., in cases of blows upon the abdomen, tight-lacing, &c., *although the teratogenetic power of external pressure is denied by most of the competent authorities.*

(b) *Pathological causes residing in the fœtus*, e.g., fœtal meningitis or encephalitis may, according to some, lead to anencephaly; fœtal arthritis is considered by some authorities to be responsible for congenital dislocations; syphilitic peritonitis may be the cause of certain intestinal atresias or occlusions, and fœtal myositis may bring about torticollis. Amniotic adhesions, however, are probably the most frequent causes of deformities

[1] See J. W. Ballantyne's article, "Teratology," in Green's "Encyclopædia and Dictionary of Medicine and Surgery," vol. ix, p. 553.

arsing during the fœtal period. According to some authorities congenital obliteration of the bile-ducts is the result of pre-natal absorption of toxin from the maternal circulation causing inflammation of the biliary passages leading to their obliteration. The author[1] has shown, however, that this theory is not altogether tenable.

We see therefore that the embryonic and fœtal periods are of enormous importance from the point of view of the welfare of the child, inasmuch as abnormalities in the external or internal configuration of the infant may arise during these periods. It is customary to call those *abnormalities that occur during the embryonic period*—and which are due to an arrest of normal development of a part of the body as the result of some chemical alteration in the composition of the maternal blood—*malformations;* whilst *those which arise during the fœtal period*, and are due to mechanical or inflammatory obliteration of a part which had already developed during the embryonic stage—are generally called *deformities*. The two classes can post-natally be distinguished histologically, since deformities show on microscopic examination evidence of tissue reaction (which malformations, of course, do not), viz., cell infiltration or fibrous tissue.

Apart, however, from the teratogenetic importance of these two periods of ante-natal life, their importance from the point of view of *ante-natal death* is very great.

We shall deal with the statistics of infantile mortality in other portions of the book (see Chapters IV and V), but in order to give the reader at this stage a practical illustration of the relative importance of the ante-natal and post-natal periods of the child's life we may briefly compare the relative mortality rates during these periods. We are all familiar with Shakespeare's seven stages of man, but these stages apply entirely to post-natal life. Physiologically, however, we may roughly divide the whole life of a human being from conception to death into several stages, viz., pre-natal, consisting of three phases—germinal, embryonic and fœtal, and post-natal, consisting of infancy, childhood, maturity and old age. Each of these is characterised by its own death-rate (per 1,000 at that period), called the *specific death-rate* for that particular stage (see p. 104). If we look at Table I and the chart in fig. 5, which gives the specific death-

[1] W. M. Feldman, "A Case of Congenital Occlusion of the Common Hepatic Duct in a Twin Baby," *Lancet*, 1924, ii, p. 113; and *British Journal of Children's Diseases*, July-September, 1925, p. 215.

TABLE I.—SPECIFIC DEATH-RATES IN VARIOUS COUNTRIES (1901-10).

Death-rate per 1,000 at age	Australia	England	Germany	Norway	Italy	United States
0—1	95·10	144·34	202·34	81·45	167·71	127·38
5	2·81	5·42	5·28	4·38	7·68	5·24
10	1·79	1·82	2·44	2·98	2·26	2·61
15	2·55	2 61	2·77	4·08	4·12	3·19
20	3·70	3·78	5·04	9·07	6·19	5·46
25	4·48	4·54	5·13	8·78	6·85	6 22
30	5·19	5 66	5·56	7·57	6 67	7·31
35	6·33	7·32	6·97	7·35	7·06	9·14
40	8·16	9·31	9·22	7·78	8·48	10·40
45	10·83	12·23	12·44	8·92	10 31	13·10
50	13 95	16·57	16·93	11·11	13·45	15·28
55	18·16	23 08	23·57	14·16	17 73	21·38
60	25·84	32·62	32·60	19·14	26·62	29·90
65	38 59	45·57	47·06	28·80	39·83	42·92
70	61·62	67·08	69·36	42·76	64·49	59·90
75	96·10	100 62	106·40	66 91	102·62	90·15

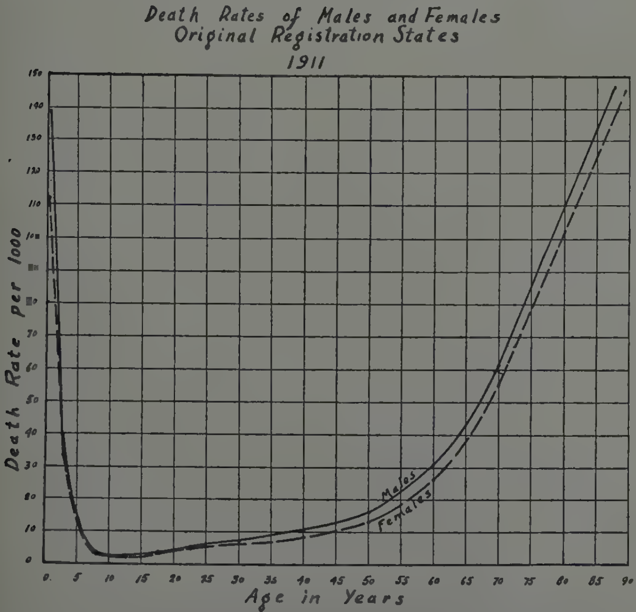


FIG. 5.—Curve giving specific death-rate for United States (after J. W. Glover).

rate from all causes at various ages from birth onwards,[1] we see that it is highest in infancy and then drops till it reaches its lowest point at about twelve years, when it begins to rise again steadily until it reaches its maximum at the end of the life span. If we confine ourselves to infancy we find that the mortality is highest in the neo-natal period (i.e., during the first month of neo-natal life). Thus in 1923 the total infantile mortality was 69 per 1,000. This means that out of every 1,000 infants that were born alive during the whole of that year, 69 died before they reached the age of one year; but out of these 69 per 1,000, no less than 32 died during the first month of post-natal life. In other words some 46 per cent. of the total infantile mortality occurred during the neo-natal period. If the infants had gone on dying at the same rate throughout the whole of the first year as during the first month, the infantile mortality for that year would have been 384 instead of 69 per 1,000. Per contra, if the rate of 69 per 1,000 had been equally distributed over the twelve months we should have expected the neo-natal death-rate to have been 5·75 per 1,000 instead of the 32 per 1,000 actually recorded.

But high as the neo-natal mortality is, the ante-natal deaths are still more abundant. In the first instance we have statistics which show that **stillbirths** occur to the extent of some 30 per 1,000 births. Secondly, there is a very large number of pregnancies that terminate prematurely with the death of the fœtus. The exact number is, in the absence of notification of abortions and miscarriages, not known with any degree of exactitude, but from figures collected by private observers we may safely say that the number is about 15 per 100 pregnancies.

This means that the number of births actually recorded in any one year represents not more than some 85 per cent. of the total number of conceptions, so that the number of pregnancies that end in abortion or miscarriage is $\frac{15}{85} = 18$ per cent. of all viable births. Hence we see that for every 1,000 live births there have occurred in 1923 (*a*) 180 abortions and miscarriages, and (*b*) 30 stillbirths, giving a total of 210 ante-natal infantile deaths, and the number of post-natal infantile deaths was 69. It is clear therefore that the proportion of ante-natal to post-natal infantile mortality is at least 3 : 1 (i.e., 210 : 69). But this is not all. For (*c*) all the deaths occurring during the early days of

[1] See J. W. Glover's "United States Life Tables," 1910, U.S. Census, 1916.

post-natal life among infants that have been prematurely born alive must really be classified under the heading of fœtal deaths. The Registrar-General's statistics for 1923 show that no less than 23 out of the 69 deaths per 1,000 were due to prematurity. Hence if we transfer these 23 from the post-natal to the ante-natal bill of deaths we find that the ante-natal : post-natal infantile death ratio becomes 233 : 46 = 5 : 1. But the toll of ante-natal deaths is even now not yet complete. The figures we have just given only take account of ante-natal deaths in the fœtal and late embryonic stages. It has, however, been shown recently that ante-natal deaths may occur at such an early stage that none of the ordinary signs of abortion are present to signalise the event. Professor Arthur Robinson recently investigated the subject in ferrets (which only ovulate after successful impregnation), and he found that by killing the females at various intervals after insemination, dead ova—both unfertilised and at various stages of development after fertilisation, including the germinal and embryonic stages—could be found lying between healthy ova.[1] Hence we conclude that the relative proportion of ante-natal to post-natal infantile deaths must be much higher even than the ratio 5 : 1 we have given above.

So much for the ante-natal : post-natal infantile death ratio. If we look at all the figures together we see that for every 1,000 viable births, no less (and probably many more) than 279 infantile lives are lost either before they are born or before they reach the post-natal age of one year. This in itself is an enormous waste of human life, but there is still the additional consideration that in the same way as ante-natal environments which are not sufficient to kill the embryo or fœtus bring about derangement of development, resulting in the production of malformed or deformed infants, so also do the conditions which bring about the death of infants, after they have been born, maim and injure those that survive; that is to say, the causes which produce infant mortality (whether ante-natal or post-natal) also produce physical degeneration (such as malnutrition, crippling due to rickets, &c.) in those that have been lucky enough to escape with their lives.

So much then for post-natal and for the intra-uterine stage of ante-natal life. But we still have to consider another most important stage of ante-natal life, viz., the *ante-conceptional* stage. Dr. Mercier's description of conception being analogous to the

[1] Arthur Robinson, *Edinburgh Medical Journal*, March and April, 1921.

addition of yeast to dough gives the truth and nothing but the truth, but it is not the whole truth, for in addition to the potential energy of development residing in the ovum and the energising activity of the spermatozoon which converts that potential energy into the kinetic kind, each of these germ cells is also endowed with latent hereditary characters which are set free in the process of fertilisation, so that the fully developed child ultimately possesses certain traits belonging to each parent. Hence we must set out still earlier in the life cycle of the child and consider the ante-conceptional stage when the germinal cells, viz., the ova and spermatozoa, which fuse together on impregnation to form the new organism, are still inside the reproductive glands of the respective parents. This means that we shall have to devote some space to the discussion of Heredity and Eugenics as well as of Puberty, for we must ever bear in mind Disraeli's saying that "The youth of a nation are the trustees of posterity."

We shall, therefore, in our study of child hygiene have to deal with the child under the following headings:—

(1) Ante-natal.
- (*a*) Ante-conceptional.
- (*b*) Post-conceptional.
  - (α) Embryonic (including germinal).
  - (β) Fœtal.

(2) Intra-natal.

(3) Post-natal.
- (*a*) Neo-natal (during first month).
- (*b*) Infancy (during first year).
- (*c*) Childhood (early, first to sixth or seventh year; late, sixth or seventh to puberty).
- (*d*) Puberty.

In this volume we shall not concern ourselves much with the hygiene of childhood and puberty, which has been dealt with in a number of books. The basis of scientific hygiene, however, is physiology, and it is only by understanding the functions and modes of working of the various systems of the body that we can have a proper appreciation of the body requirements, and are thus able to formulate those "laws of health" which constitute the science and art of hygiene. Hence in our consideration of the various portions of child hygiene we shall have to discuss briefly the main principles of ante-natal and post-natal child physiology. But before starting on our main theme we shall give an account of the history of the subjects from the earliest times to the present day.

CHAPTER II

THE HISTORY AND DEVELOPMENT OF CHILD HYGIENE

"There is no part of history so generally useful as that which relates the progress of the human mind, the gradual improvement of reason, the successive advances of science, the vicissitudes of learning and ignorance which are the light and darkness of thinking beings, the extinction and resuscitation of arts and the revolution of the intellectual world."

JOHNSON.

IT is a pleasant and instructive pastime to study the historical development of any branch of art or science, and to trace its gradual evolution from its dark and primitive state to one of light and refinement. It is pleasant because it affords the student an intellectual exercise as good and as keen as any that we can think of. "A mouldering medal is a letter of twenty centuries. . . . The painting round a vase, the scribble on walls . . the point of an epigram—each possesses its own interest and value," for they provide one sufficient entertainment and scope for pleasant cogitation for many an idle hour. The books of ages past contain the recorded wisdom and experience of numerous intellectual giants of days gone by, and by studying their contents we acquire "the experience of age without either the infirmities or inconveniences thereof." It is instructive because it illustrates the oft-quoted saying of King Solomon that "there is nothing new under the sun"—not even the recently re-discovered sunlight treatment. For as we shall see in what follows, the principles of child hygiene have changed very little from the times of remote antiquity. Not indeed that fashions have not changed, for, in the words of Robert Hutchison, "there is a fashion in the cut of the robe of Æsculapius just as much as in that of more mundane garments"; but what has taken place in the art and science of our subject during the thousands of years under our survey is a gradual transition from a state of empiricism to one of scientific reasoning. The historical study of child hygiene should both humble and uplift us at one and the same time, for it will show us that whilst we have not

achieved any marked success in the discovery of new gems, we have succeeded in purifying and polishing the old ones as well as in extracting and refining the precious metals from the rough ores that have been handed on to us by our ancestors.

In the case of our subject, the development of which we shall consider from its scientific as well as its sociological aspects, we need not look for "sermons in stones," for fortunately the vast hoards of knowledge of ancient philosophers have in most cases been bequeathed to us in the form of written records so that he who runs may read.

Religion and Prosperity in Relation to Child Protection.

These records of the past teach us that the care and attention devoted to infants and children, even amongst races which occupied a high position in the scale of civilisation, depended

FIG. 6.—The Egyptian Goddess Isis suckling Horus.

upon two factors, viz., the type of their religion and the constitution of the soil upon which they lived. People who lived on fertile soil, so that the food supply was good, and whose presiding deity was of the matriarchal type—as represented by a mother goddess (figs. 6, 7), treated their children with sympathy and kindness.

On the other hand, amongst peoples who lived on barren soil, where the struggle for existence was great, and whose deity was of the patriarchal type—as represented by a male god—the

FIG. 7.—The Goddess Earth, or Terra Mater. Frieze of left side of west entrance of the *Ara Pacis* (Altar of Peace) erected in the Field of Mars by the Senate to commemorate the then recent and victorious return of Augustus to Rome, from a double campaign in Spain and in Gaul in the year 13 B.C. This allegorical relief, which is preserved in the Uffizi of Florence, shows a beautiful woman holding two children, as well as grapes and other fruit, on her lap. On the left is a woman riding on a swan (representing the genius, Air) and on the right is another woman riding on a sea-monster (representing the genius, Water)—the two fertilizing genii of the earth. Below are trees, flowers and animals.

FIG. 8.—Picture on a Babylonian cylinder showing the sacrifice of a child to a male god. (From the *Jewish Encyclopædia.* After Menant.)

reverse conditions prevailed; the infant was either murdered at birth, cruelly maltreated during its early infancy and childhood, or sacrificed for ritual reasons (fig. 8).

In some parts of the world like China and India, where the economic conditions have always been bad, and where the religion was of the patriarchal type, *infanticide*, especially of female babies, was, and still is, quite common, in spite of numerous edicts issued from time to time against such a practice. In China, for instance, the first official decree against infanticide was promulgated by Choen Tche, in 1659, in which he said: "I have heard that the sad cry uttered by these girl-babies as they are plunged into a vase of water and drowned is inexpressible. Alas, that the heart of the father or mother should be so cruel." Since then several similar decrees have been issued—the last being as recently as 1848, but the practice still continues. It is estimated that some 30 or 40 per cent. of new-born female infants are still being drowned in China every year. Among the Aryans in Ancient India, male children were valued for the purpose of raising an army, but females were preserved in numbers sufficient only for breeding soldiers and no more. As late as 1871 a last endeavour to suppress this 5,000 years old practice was made by the Indian Government when "the Infanticide Act" was passed. Even in such a progressive country as Japan, infanticide for economic reasons is said to have been common after the Russo-Japanese war, in the early years of this century.[1] Nearly in the whole of Greece and in Rome until the early years of the present era, infanticide, abandonment of infants, and deliberate inductions of abortion were the order of the day. The same was the case with the ancient Persians, Carthaginians, Arabs and Phœnicians. The only people who did not practise these abominations were the Israelites, and to a certain extent also the Egyptians. But outside Judaism, although efforts to stop infanticide and child neglect were made even by pagan emperors and statesmen (see pp. 41-43), it is only with the advent of Christianity, as well as the preaching of Mohammedanism, that these horrors ceased. Tacitus in his great hatred for Jews speaks sneeringly of their practice not to allow new-born babies to be put to death ("Necare quemquam ex agnatis nefas putant," "Histories," book v, sect. 5).

[1] The best illustration of the importance of the economic factor or the condition of the soil in relation to infanticide is afforded by an official statement from Japan (in reply to an inquiry by G. H. Payne) to the effect that in a certain part of that country where the soil was barren and infanticide was common, a wise governor, instead of punishing the offenders, established an efficient system of land irrigation, with the result that not only did infanticide cease, but the average size of the family was considerably increased.

Infanticide has remained up to the present day amongst the savages, e.g., the South Australians, Kaffirs, West African negroes, Polynesians, Papuans, &c., as well as amongst certain civilized nations, such as the Chinese, &c., as we have seen above.[1]

But notwithstanding the brutal indifference displayed by nations of antiquity in the treatment of their infants, we have incontrovertible evidence to the effect that serious efforts were made by thoughtful men and women at different periods in the history of the world to instil into the minds of the people the economic and national value of child life, and to instruct the general public in the fundamental principles of child hygiene. As we shall see, however, it is not till comparatively recent times that the constant falling of this gentle drop succeeded in breaking the dense rock of apathy and indifference.

Babylon.

During the **Assyro-Babylonian period,** i.e., about 3000 B.C., the type of religion which, in the older Sumerian culture, had been patriarchal, became matriarchal. The mother-goddess, Ishtar, was the presiding deity, and hence we find in the Code of Hammurabi (about 2500 B.C.) the oldest codification of laws known, and one which was based upon centuries of tradition, strong evidence of kindness bestowed upon children. Any assault on a pregnant woman resulting in miscarriage was punishable by a fine of ten silver shekels. Similar protection was, as we shall see, afforded to an expectant mother by the Mosaic law, which no doubt adopted much that was good from the Babylonian culture in the atmosphere of which the Hebrews lived. Orphans as well as children of divorced women were well provided for, and liberal allowances were made for adopted children. A foster-mother through whose negligence a baby, entrusted to her care, died, had to forfeit one of her breasts. Widows or deserted mothers were given pensions by the State.

Egypt.

In ancient Egypt, too, the religion was of the matriarchal type and the land was rich and productive. Hence, as we might expect, infanticide was almost unknown and the children were

[1] Mary Slessor (1848-1915) did a great deal amongst the savage natives to discourage the practice of infanticide.

treated with sympathy.[1] Several *Papyri* have been discovered which record some of the medical lore of the Egyptians, but the *Papyrus Ebers*, discovered by the German Egyptologist, Georg Moritz Ebers, in 1875, constitutes practically a "system" of medicine written about 1550 B.C., and contains several details regarding the care of infants, such as advice about breast-feeding, and methods of determining the flow of milk to a nursing woman's breasts. The treatment is also described of several different infantile ailments such as digestive troubles, skin diseases, worms, &c. During the Ptolemaic period (330 to 43 B.C.),[2] maternal breast-feeding fell into disuse and was substituted by wet nursing. The foster-mother was under contract to suckle the infant during the first six months, and to give it "the best cow's milk"—of which there was a regular daily delivery—for

Thebes.

FIG. 9.—Women carrying their children in a funeral procession. (Wilkinson, "The Ancient Egyptians.")

another eighteen months. There were special sucking-bottles, a sample of which is to be found in the museum at Cairo; and a relief in the Louvre unearthed from the ruins of the palace of King Sardanapalus of Nineveh, who lived in the ninth century B.C., shows a mother and a baby with a feeding bottle (see fig. 14A on p. 40). The infant was carried about in loose clothing—as opposed

[1] The decree of killing male infants at birth recorded in the Bible (Exod. i. 16) only applied to Hebrew infants, because the Egyptians were afraid of the growing strength of the Hebrew population. The midwives, however, disregarded this decree (*ibid.* 17).

[2] Ptolemy I was one of the generals of Alexander the Great, of Macedon, who became King of Egypt in 323 B.C. He was a friend of Euclid and founded the museum and library at Alexandria. His son, Ptolemy II, patronised learning, and made the library and museum a centre of literature and science. The Septuagint (the Greek translation of the Old Testament) is said to have been made by his order. The last of the Ptolemys was Ptolemy XIII, who was put to death by Cleopatra.

to swaddling clothes—and allowed to live a wholesome life in the open air. According to Aristotle, Egyptian women had many children, all of whom were treated well, and according to Herodotus

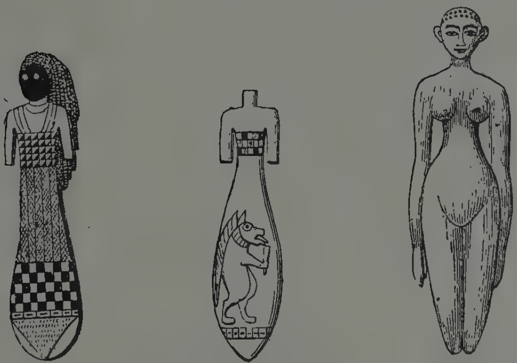
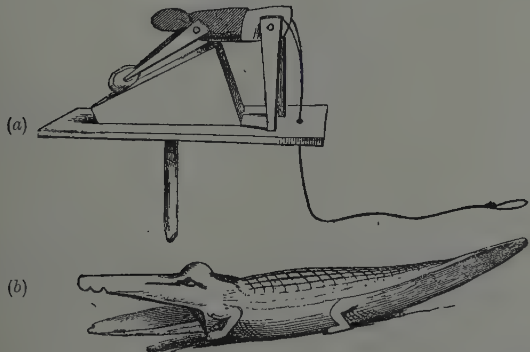


FIG. 10.—Wooden dolls. (Wilkinson, "The Ancient Egyptians.")



Leyden Museum.

FIG. 11.—Children's toys. (Wilkinson, "The Ancient Egyptians.") (*a*) A man doing some work. The model was made to work by pulling a string. (*b*) Crocodile.

Egyptian children (as well as adults) had their hair shaved (except for side locks) and were allowed to go out in the sun with the head uncovered. This custom particularly applied to the

lower classes, and Herodotus attributes the hardness of the bones of the head and the lack of baldness amongst Egyptians to this custom.

The sympathy shown by Egyptian midwives to the babies at whose birth they assisted is well exemplified in Exodus, in connection with the birth of Moses.

From designs on Egyptian monuments we learn that the infants were carried by their mothers, either on the back or in front of the abdomen, and were kept in place by a piece of material arranged in the form of a pouch (fig. 9). Dolls and toys were given to the infants to play with (figs. 10 and 11).

The Egyptian girls married very young, mostly between 12 and 14 years of age, but Ebers found a record of a case of a 9 years' old mother.

FIG. 12. — Maimonides, physician, philosopher and mathematician, concerned himself also with preventive medicine. He writes that "the purpose of medicine is to teach the cause of health, the correct dietetic hygiene, and how to prevent disease." He expressed his disbelief in anything which cannot be attested by (1) the same logical proof as in mathematics, or (2) the perception of the senses (eyes, ears, &c.), or (3) *unimpeachable* authority. A man, he says, must never cast his own judgment behind him; "the eyes are set in front, not in the back."

Ancient and Mediæval Hebrews.

That great care was bestowed upon children by the ancient Israelites is evidenced by the numerous references to the welfare of children during the various stages of their ante- and post-natal development, which are widely scattered about in the various books of the Bible (2000 to 450 B.C.), the Talmud (450 B.C. to 500 C.E.), Midrash (sixth to ninth centuries), Maimonides (fig. 12)

(twelfth century), Joseph Karo (sixteenth century), and later Jewish writings. Children of either sex were, and still are, considered by Jews as the greatest blessing God could bestow upon man. Children being "an heritage of the Lord" (Ps. cxxvii. 3) it is obvious that such a legacy must be carefully looked after. Permanent celibacy was characterised by a Talmudic Rabbi as being akin to murder. "Every Jew," says a Rabbi, "who refrains from reproduction is like one who sheds blood." Contraception was forbidden for similar reasons—except in very special cases, such as extreme youth or illness of the wife and in the case of a nursing mother. Male children were, and are, specially favoured. The Talmud contains a good many statements which are of real **eugenic** value. "A girl of good pedigree, even if she be poor and an orphan, is worthy to become the wife of a king." Mercenary marriages were discouraged. The marriage of the physically unfit, or of those with a family history of epilepsy or other transmissible disease, is looked upon by the Rabbis with disfavour, since similar illnesses may occur in the offspring.

Ante-natal Care.—There is much excellent advice in the Hebrew literature regarding the **care of the expectant mother,** from the point of view of her unborn baby. A pregnant woman is to be sheltered from fright, bad news, unpleasant odours, &c., because such are likely to bring on premature labour. For a similar reason she is not to undergo undue fatigue, especially on a hot day, must not have a hot bath, and must avoid drastic purgatives. The protection of a pregnant woman against assault was specifically legislated for in the Bible: "If men strive and hurt a woman with child, so that her fruit depart from her and yet no other mischief follow, he shall be surely punished according as the woman's husband will lay upon him and he shall pay as the judges determine" (Exod. xxi. 22). Similar protection was afforded by the Babylonians, as recorded in the Code of Hammurabi. Induction of abortion is an offence punishable by a fine, although according to Josephus it is to be considered as murder. As a protection against miscarriage pregnant women used to wear an amulet called *eben t'kouma*, or stone of preservation. The popular belief in the influence of *maternal impressions* dates of course from the time of the Bible: "Jacob took him rods of green poplar . . . and pilled white strakes in them . . . and the flocks conceived before the rods and brought forth cattle ring-straked, speckled and spotted" (Gen. xxx. 37-39).

Similar anecdotes are found in the Talmud and Midrash (600-900 C.E.). But whilst in Hebrew literature we find the belief that maternal impressions either during conception or immediately preceding it exercised an influence upon the future child, there is never any mention there of the effect of such impressions upon the unborn infant during pregnancy, although Greek and Roman writers refer to it. In this respect, as we shall see (Chapter IX), the Hebrew writers were more scientifically correct than their contemporaries. Directions are given regarding the food that an expectant mother should eat and that which she should avoid. Meat, fish, parsley, coriander, "paradise apples" (? oranges), and in ordinary cases wine are particularly recommended as giving rise to healthy, strong, and beautiful children. This, as we now know, is a diet rich in vitamins which are so necessary for the fœtus. On the other hand, certain indigestible articles of diet, as well as in special cases (see Judg. xiii. 4, and Luke i. 15) alcoholic beverages, were forbidden.

Parental alcoholism was believed to influence the future of the child, whether the indulgence was before or during conception. A certain Rabbi, Nachman, remarked that the daughters of another Rabbi, Bibi, required massage and cosmetics to make them beautiful because their father indulged in wine, but his own daughters did not require any artificial means of beautification because he was an abstainer. Again, the Talmud enumerates a number of things which have an influence during conception upon the future child, and mentions a state of intoxication as one of them (cf. Chapter IV, p. 114). Intra-uterine pressure was believed to be a cause of monstrous birth (see Chapter I, p. 7).

Intra-natal Care.—In cases of difficult labour, when the question arises between saving the life of the mother or that of the fœtus, the latter must be sacrificed in favour of the former. If, however, the greater part of the infant has already been delivered, then the infant has the same legal right as the mother, and nature must be left to take its course—if it is impossible to do anything to save both mother and child. Cæsarean section, however, is discussed in the Talmud, both in the case of living mothers as well as in those who died in labour, in whom the operation was performed to save the child. The fœtus was, according to an old law, considered a separate being, and therefore in a case of sentence of death on a pregnant woman, the execution had to be postponed until after labour; but later legislators recognized the fœtus as only a part of its mother, and it therefore had to share her fate in a case of capital punishment.

POST-NATAL CARE

(*a*) **Neo-natal.**—To resuscitate an apparently stillborn baby several remedies are given, but insufflation of air by means of a fan is specially recommended, as is also a method of swinging the baby, similar to the modern Schultze method; though friction and massage of the child with the *unseparated* placenta (by means of which more blood would be transmitted to the infant) is also mentioned. The cord was ligatured and cut by means of a stone and the body was salted, anointed with oil and, contrary to what one might expect from the stay of the Israelites in Egypt, but in accordance with Greek and Roman customs, was wrapped in swaddling clothes ("to straighten out the limbs which were compressed during delivery"), a custom which still prevails among the less enlightened Jews in certain parts of Eastern Europe. The salting of the baby was quite a common custom among the various nations at that time and was probably used for its antiseptic and stimulating properties. Soranus, as we shall see (p. 33), recommends it, and Galen orders it "for the purpose of rendering the skin harder and more solid." A good description of a neglected new-born infant is given in Ezekiel (xvi. 4-9). "In the day thou wast born thy navel was not cut, neither wast thou washed in water to supple thee; thou wast not salted at all, nor swaddled at all. . . . And when I passed by thee I saw thee polluted in thine own blood. . . . Then washed I thee with water . . . and I anointed thee with oil." Some mothers bathed their children in wine—a practice probably adopted from the Greeks (see p. 33). Another custom, also probably copied from the Greeks, was pressure of the head to mould it into shape. Both Hippocrates and Moschion (pupil of Soranus) speak about it. Circumcision—an operation which every Jewish male child has to go through on the eighth day—must, according to the Talmud, be postponed if the child shows the slightest trace of ill-health, e.g., pyrexia, jaundice, &c.; and if there is any history of hæmophilia in the family the operation must not be done at all.

Infanticide was, of course, punished by death, although we find a couple of instances in the Bible of actual or attempted ritual sacrifices of the first-born sons. Thus we read (2 Kings iii) that when Mesha, king of Moab, found that "the battle was sore for him . . . he took his eldest son that should have reigned in his stead and offered him for a burnt-offering upon the wall." But the verse continues, "And there was a great

indignation against Israel, and they departed from him." The story of the aborted sacrifice of Isaac (Gen. xxii) is another illustration of similar practice, but the Bible makes it clear that it was merely a test imposed by God to try Abraham's faith. There is still a reminiscence of this rite among the present-day Jews in the ceremony of the "Redemption of the first-born son" (*Pidyon Habén*) which is carried out when the child is thirty days old. The father presents the child to a Cohen (a reputed descendant of Aaron the High Priest, and who has the religious right of possession of the child), and says: "This my first-born son is the first-born of his mother, and the Holy One, blessed be He, hath given command to redeem him" (Num. xviii. 15-16). After some parley between the Cohen and the father, the latter gives the former a stipulated sum of money, viz., five shekels (about fifteen shillings), saying, "I desire to redeem my son, and here thou hast the value of his redemption."

But, although infanticide of a full-term baby was a capital offence, the killing of a *premature* infant under thirty days old, i.e., before one could be sure of its viability, was considered an offence punishable by a fine only, and was placed in the same category as an abortion. It is interesting to note that in agreement with the views of Hippocrates, Pliny, Galen, and Soranus (see p. 32), but in opposition to the opinion of Aristotle, an eight months' baby was considered less viable than one born at seven months. The length of a new-born baby is given in the Midrash as a little more than a cubit, which would correspond to about twenty inches.

Foundlings were, in Talmudic times, to be cared for by the State, and children that were born with malformations, which amongst the Romans would have been thrown into the Tiber, were treated by expert surgeons.

(*b*) **Later Infancy.**—Maternal breast-feeding of her baby was an obligatory duty which every woman owed to her husband unless she happened to have twins, when it was the husband's duty to provide a nurse for one, whilst she suckled the other. If the woman was very rich, she was allowed to procure a wet-nurse, except in the case of a delicate infant. Breast-feeding was continued for two years, and during lactation a wife was permitted to use contraceptive measures, lest the intervention of a pregnancy would interfere with the supply of milk. For similar reasons a widowed nursing woman was not allowed to remarry before the expiration of two years from the birth of her

baby. This held good, even if the baby died in the meantime, as a precaution against a possible temptation to get rid of her baby in order to remarry. On the other hand, a bereaved husband was, under similar circumstances, allowed to remarry after the expiration of the seven days of mourning, in order that the baby might have somebody to look after it.

Jeremiah gives a poetic description of a mother who refuses to nurse her baby: "Even the sea-monsters draw out the breast, they give suck to their young ones; the daughter of my people is become cruel, like the ostriches in the wilderness. The tongue of the sucking-child cleaveth to the roof of his mouth for thirst" (Lam. iv. 3, 4). Orphaned babies used to be carried about from house to house to nursing mothers who, out of compassion, gave them suck. According to Jewish law (Deut. xxiv. 19-21) a land-owner must, during the harvest season, allow the poor to come and collect what was left after the first gleaning, or what was forgotten to be removed. The early morning was reserved for that purpose for poor nursing mothers; midday was reserved for children, in order that they may not be deprived of their morning sleep.

Hygiene of a Nursing Mother.—A nursing woman was given a more liberal diet and less work to do. She was also recommended to have a reasonable, but not an excessive, supply of wine, as small quantities of wine were believed to promote the flow of milk. Also certain articles of diet, such as meat, parsley, citrons and coriander, were particularly prescribed as having a beneficial influence on the appearance and physique of the child (such a diet would be nutritious and rich in vitamins), whilst others, such as mustard, cress, salt fish, pumpkin, unripe figs, vinegar, &c., owing to their deleterious effect upon the infant, were forbidden her.

Diet after Weaning.—When breast-feeding ceased the child's diet consisted of eggs, butter, milk and honey, and later, nuts, meat and wine. Maimonides discourages the use of alcohol by children and adolescents.

The Child's Sleep. — The child, in Talmudic times, had a separate cot which had short legs fixed to the floor for safety. The cots were made either of wood or of glass. Swinging cradles were also used. These had little bells attached to them, whose monotonous sound produced by the swinging helped to induce sleep. A piece of waterproof material was placed under the child to keep the bedclothes clean. A fan was used to keep

the flies away from the sleeping baby. The child was never left alone in its cradle by day or by night, and for this reason the cot was only used in the daytime; at night the baby was taken into its mother's bed. Cases of overlaying necessarily occurred; one such case is described in connection with the Judgment of Solomon: "And the woman's child died in the night because she overlaid it" (1 Kings iii. 19). Indeed the Bible does not mention cots or cradles anywhere. It is probable that during the day the mothers in Biblical times carried their infants about with them like the Egyptians (see fig. 9, p. 18).

The subject of **infant mortality** also engaged the attention of the Rabbis. That the death-rate of babies was high is evidenced from the mournful exclamation of a Rabbi that "it is easier to see a whole forest of trees grow up than to rear one baby in Palestine" (Midrash). The **causes of death,** some of which are of scientific interest, are mentioned, e.g., *ascara* (i.e., croup), contamination of water (which was also believed to be a cause of abortion), spread of infection by means of flies, &c. In the time of the Temple two days a week were ordained as fast days, viz., Tuesday and Thursday, the former to forestall the dreaded *ascara* (croup), the latter to protect pregnant women and nurslings from untoward accidents. Certain substances were applied to the baby's head to keep away insects. When the infant sat up a waterproof bib was tied round its neck to protect it against the heat and scratches by cats.

When the child reached school age, i.e., six years, it was, in Talmudic times, medically examined to ascertain its physical fitness to attend school; if it was not strong enough, schooling was postponed for another year. The child was washed and cleaned and taken to and from school by its mother or eldest sister. The school buildings of that time were well lighted and well ventilated, and were provided with open-air playgrounds and other sanitary appliances. *Open-air schools* are also mentioned in the Talmud. The children were taught various physical exercises, including swimming, and were specially guarded against immoral practices. It was the duty of the father to provide for his children until they reached the age of puberty. Poor children were cared for by the State.

Illegitimate Children.—The term "illegitimate" has a different sense in Hebrew law and literature from that which it has in English law. An illegitimate child, or *mamzer*, is one that has been born of any marriage prohibited by the Bible (see

Lev. xviii). A child born extra-maritally is not a *mamzer* and does not legally suffer the disqualifications appertaining to illegitimacy.

If during her husband's absence a wife misconducts herself with another man but returns to live with her husband, the offspring is legitimate. Also the child of a betrothed woman is legitimate if her affianced husband does not deny paternity. The principal disqualification of a *mamzer* is that such a child may not marry into the community.

Brahminian Medicine.[1]

In the literature of ancient India, of which the work of Susruta (second century B.C.) can be taken as the type, we find a great deal of space devoted to eugenics, the care of the expectant and parturient mother, as well as to the hygiene of the infant. In the choice of a wife care should be taken to avoid families with histories of consumption, epilepsy, &c. The marriage age for men was 25 and for girls 12. Children of very young mothers are stated to die before birth or to be born weaklings. Fœtal death may also occur as the result of worry or disease of the mother. An eight months' baby is not considered viable.

The nutrition of the fœtus occurs through the cord, which "without doubt carries the essence of food from the mother to the fœtus." Details are given regarding suitable diets at various stages of pregnancy, and sanitary, hygienic and well-equipped "lying-in" homes are described where women were sent at the beginning of the ninth month of pregnancy; these were well staffed with doctors, midwives and nurses. Certain rules are given regarding the *immediate care of the new-born infant.* The cord was tied and cut eight fingers' breadths from the abdomen, and cold water was sprinkled on the baby's face to stimulate respiration. The baby and mother were washed immediately after birth—rich children in water in which gold or silver had been previously heated. The technique of breast-feeding is described, and minute details are enumerated in connection with the choice of a wet-nurse. She must be neither too young nor too old, of medium build, and of good health and moral character. Her breasts must be of the proper shape and size and the nipples must be properly formed. When breast feeding is impossible,

[1] See Heinrich Joachim, "Die Diätetik und die Krankheiten des Kindlichen Alters bei den alten Indern," *Arch. f. Kinderheilk.*, xii, 1891, p. 179.

goat's milk should be given, or, if the latter is unobtainable, cow's milk. The infant must be handled with the greatest gentleness, and is not to be suddenly roused from its sleep. It must not be made to sit up prematurely for fear of causing spinal curvature. It is to be sheltered from inclement weather, from the glare of the sun and from contaminated air. The physical properties of good milk are described. Post-natal child life was divided into three periods, viz.: (1) Infancy, from birth to the end of the first year; (2) second period, from 1 to 3 years, when the child was fed on milk and rice; (3) third period, from 3 to 15 years.

Greek and Roman Medicine.

When we come to Greek and Roman literature we find references in Galen, Quintillian, Lycurgus, Pliny and others to the effects of maternal impression. In the Homeric period (950 B.C.), as evidenced by references in the Iliad and the Odyssey, we find that infants, when not killed at birth, were breast-fed by their mothers, except in the case of the upper classes who employed foster-mothers; e.g., Hecuba nursed Hector, Penelope breast-fed Telemachus, &c. The law of Lycurgus required Spartan mothers to look upon statues of Castor and Pollux in order that they might give birth to strong and beautiful children. The new-born baby was given a warm bath and wrapped in swaddling clothes. It was provided with a cradle and given plenty of toys. In Sparta children were reared on the principle of the "survival of the fittest." According to the laws of Lycurgus (880 B.C.) a puny or ill-formed child was neglected, since it was "neither for the good of the child itself nor for the public interest that it should be reared." The children at birth were plunged into a bath of iced water and were afterwards left naked. Those that survived were considered fit to be reared! Soranus, as well as Galen, deprecates this Spartan and Germanic custom of immersing the babes in cold water. Claude Quillet, a French physician and poet of the seventeenth century, speaks of this Germanic custom as follows:—

"... so barb'rous were their Ways, 'tis said,
They snatched the Infant from the Mother's Bed:
And lest it should in Hardiness decline,
Plunged it yet reeking in the Frozen Rhine.
Their Force on Nature was not less extream,

Than when red Iron's flung into the stream.
They taught 'em, from their Childhood to defie
The Frosts and Colds on an inclement skie.
Thus hard, like Beasts, their humane Limbs they made
Nor were of Weather, nor of Toil afraid.
Such sure as cou'd this horrid Bath survive
Must from Caucasian Rocks their Birth derive."

Swaddling clothes were not used but the child's limbs were allowed to move about freely. "Spartanorum autem infantes in lucem editi fasciis non illigabantur sed soluti et nudi ferebantur: Arte tamen quadam membra eorum ad concinnitatem et decus conformabantur" (Th. Bartholin, quoted by Bokay). The education of boys was mainly physical, such as running and boxing. They were flogged to teach them endurance. Girls were also hardened by means of various exercises with a definite eugenic aim, viz., that, as Plutarch in his "Life of Lycurgus" says, "the fruit wherewith they might afterwards be conceived, taking nourishment of a strong and lusty body should shoot out and spread the better." Maternal breast-feeding was ordained, and such was the respect paid to a nursing mother that people had to salute her in the street and make way for her. Celibacy was penalised and fertility rewarded. The man with three children was excused night duty, and he who was the father of four was exempt from taxation. In the time of Xenophon the laws of Lycurgus were disregarded, and in spite of the efforts of later legislators the Lycurgian discipline fell into disrepute (Roper). Plato considered the fœtus as an animal which could therefore be killed, and Quintillian goes as far as to say that infanticide is sometimes a real act of kindness (compare p. 31).

Suetonius writes that the Senate, alarmed by a prophecy that in a certain year a child would be born who would conquer the Roman Empire, ordered all male infants born during that year to be slain—an edict analogous to that issued by the King of Egypt as a precautionary measure in relation to Hebrew male children, as recorded in Exodus, chapter i. But in the same way as the Egyptian midwives refused to enforce Pharaoh's decree, so also was the Roman edict defeated by the fathers, who secretly preserved their sons, each father dreaming that perhaps his own son was destined to become Emperor of Rome. (Compare also the legend regarding Remus and Romulus.)

Many references to the hygiene of infancy are also found in the writings of Hippocrates (460 to 370 B.C.). The "Father of Medicine" may also be called the Father of Pædiatrics (fig. 13).

Indeed, legend narrates that over his grave, between Gyrto and Larissa, a swarm of bees settled itself, and their honey was used as a cure against aphthous stomatitis of infants. He records the observation that œdema during pregnancy is likely to result either in a dead fœtus or in a debilitated infant.[1] He further knew that the mortality of infants increased when they were taken off the breast. Hippocrates, however, devoted himself more to the pathology than to the hygiene of early life.

Many Greek physicians in the early part of the Christian era followed in the footsteps of Hippocrates, among them being Celsus (first century), Soranus of Ephesus (second century), Galen (second century), Oribasius, a pupil of Soranus, Aetius (sixth century), and Paul of Ægina (seventh century).

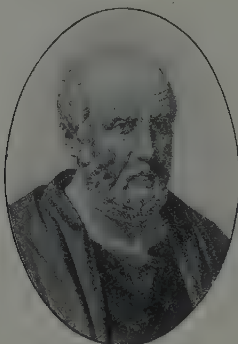


Fig. 13.—Hippocrates

Soranus of Ephesus, the Greek physician who flourished in Rome during the reigns of Trajan and Hadrian in the second century A.D., may rightly be called the first practical obstetrician, pædiatrician and child hygienist in antiquity. His book, consisting of sixty-six chapters and entitled *περι γυναικείων παθων*, deals very fully with pregnancy, labour, hygiene of infancy, pædiatrics and gynæcology, and is remarkable for the fact that the chapters dealing with infancy contain a most comprehensive systematic account of the hygiene of early life. Indeed, it is so complete and sound that it may with a few modifications serve as a textbook on the subject of infant hygiene at the present day.

[1] Quoted by Traugott Kroner, *Jahrb. f. Kinderheilk.*, x, 1876, p. 340, and xi, 1877, p. 83.

In the following few pages I shall give a brief summary of those chapters of the book which deal with fœtal and infant hygiene,[1] and briefly compare his teaching with that of his contemporaries or predecessors as well as with modern knowledge.

But before giving an abstract of Soranus' work it is of some interest to consider briefly the views held by the ancient Greek and Roman philosophers on the subject of **heredity and eugenics.** Euripides says, with more rhetoric than truth: "Is it not wonderful that poor soil, blessed with a favourable season, bears corn in abundance, yet with human kind the worthless is always base, the noble never anything but noble. . . . No education can transform the bad child of evil stock." The Greek poet, Theognis, of Megara (sixth century B.C.), deplores the evils of mercenary marriages as follows: "We seek well-bred rams and sheep and horses and wish to breed from them. Yet a good man is willing to marry an evil wife, if she brings him riches. . . Wealth has confused the race. Marvel not that the stock of our folk is tarnished, for the good is mingling with the base." Plato, a century later, proposed State intervention to regulate marriage by means of *ante-nuptial medical examination*, so that the best should mate with the best and be encouraged to have large families, which should be reared by the State, whilst the inferior stock should mate with people of the same level and their children be "put away in some mysterious unknown place, as they should be." Aristotle also advocated State regulation of marriage.

In addition, therefore, to the economic and religious reasons for child murder, infanticide was also practised in Greece and Rome for eugenic reasons. Lycurgus, Plato, and others, as we have seen, considered infanticide necessary in the interests of the State. Seneca[2] wrote: "We bludgeon mad dogs . . . we slay diseased sheep, lest they may infect the flock. We stifle infant monsters, we drown the weakly or deformed new-born. It is not from cruelty, but from common sense that we separate healthy stock from those that might corrupt them." The Bœotians, who condemned infanticide, were considered by the rest of Greece as "a rude, uncultivated and barbarous people."

[1] The text used for the purpose of this abstract is the Latin translation (1869) by Franciscus Zaccharias Emmerius, entitled, "Sorani Ephesii, Liber de Mulieribus Affectionibus. Traiecti ad Rhenum apud Heminx et filium. CIↃIↃCCCLXIX."

[2] "De Ira," i, 15.

Soranus starts his book with the consideration of the requisites of a good midwife. She must be strong, patient, fond of work at any time by day or night, of reasonable education so that she can profit by reading books dealing with her profession, and must not be fond of money. She must also have properly shaped fingers.

In Chapter X he deals with conception. He discusses the most favourable time for conception—which he believes to be immediately after a menstrual flow—and speaks of the influence of *maternal impressions*, during conception, upon the child. "Ape-like children," he says, "have been born to women who during conception looked at monkeys" (Quo modo quaedam in amplexu simiis conspectis similes simiis fœtus gestarunt in utero). The diagnosis of the sex of the fœtus is considered in another chapter (Chapter XIII). This is based upon the vigour of the fœtal movements, which are stronger in the case of a male child.

Ante-natal care is discussed in Chapters XIV-XXI. Advice is given regarding the expectant mother's diet, and mode of life. Hark work, purgatives and excessive marital relationships are to be avoided. The question of contraception is considered in detail in Chapter XVII.

Chapter XXV. The **signs of viability** of the new-born infant are: (1) Good health of the mother during the pregnancy. (2) Conceptional age of the child, either nine months or seven months, but not eight months. (3) Lusty cry immediately after birth. (4) Perfect formation of body, such as patency of the œsophagus, anus, urethra, &c. (5) Free kicking of limbs. (6) The presence of normal sensibility, such as can be ascertained by pressing the finger in different parts of the body.

With the exception of the first two signs his method of judging the viability of an infant is perfectly correct. We now know, of course, that apart from certain conditions of the mother, such as syphilis, active tuberculosis, toxæmia of pregnancy, &c., the health of the fœtus is almost entirely independent of that of the mother (see p. 122 *et seq.*). Indeed, even the contemporaries of Soranus were aware of this fact. For, in the seventh century B.C., the Roman Emperor, Numa Pompilius, decreed that no woman dying during pregnancy should be buried before her unborn infant had been removed from her with the object of saving its life. "Negat lex regia muliebrem, quae praegnans mortua sit, humari, antequam partus ei excidatur: qui contra fecerit spem animantis cum gravida peremisse videtur" (Digest 1,

xi. c. 8, quoted by Kroner, l.c.). We have also seen that post-mortem Cæsarean section was done by the Jews to save the unborn infant. Aristotle was aware of the viability of an eight months' fœtus.

Chapter XXVI.—After the lapse of a short interval to enable the infant to recuperate from the traumatism of birth, the *cord is tied* in two places and *cut* by means of a sharp instrument between the two ligatures, to prevent fœtal and maternal bleeding, at a distance of four fingers' breadths from the abdomen. The cut cord is dressed with wool or cotton-wool. The cautery or any instrument other than knife or scissors (i.e., sharp instruments) is not to be used on account of the pain and inflammation it is liable to cause.

The chapter continues with the *baby's bath*. Soranus condemns the method adopted by "some barbarians, Germans and Scythians, and even some Grecians," of plunging the baby, immediately after section of the cord, into cold water for the purpose of hardening it. Such treatment may either kill the baby or render it liable to convulsions or apoplexy.

Galen also speaks strongly against this brutal practice, and we now know, of course, that the thermotactic apparatus of a new-born infant is so feebly developed that any undue exposure to cold is liable to kill it.

Soranus further deprecates the bathing of the infant in wine (compare method of Hebrews, p. 23), and speaks about the removal of the vernix caseosa.

Chapter XXVII deals with the *sprinkling* of the baby *with salt*. He warns against the danger of allowing salt to come into contact with the baby's eyes or mouth. The salt is to be washed away with tepid water, and, with the aid of a finger, the *nose, mouth* and *ears are cleaned*. A little *oil* is to be *instilled* into the eyes in order to get rid of any thick discharge which might otherwise affect the sight.[1] This is a most remarkable statement, since it practically forestalls the Credé method of silver nitrate instillation to prevent ophthalmia neonatorum.

After the bath the little finger with the nail properly trimmed is to be inserted into the anus to clear it of meconium, as well as to remove any membranous occlusion of the anus.

In Chapter XXVIII Soranus condemns the Thessalian method

[1] Etiam oculis infundere oportet oleum, nam utile est de iis abstergere crassissimum, qui iis inest, humorem; quod nisi fiat, plerumque infantes dum enutriuntur visus hebetudine laborant.

of tying up the infant in a wooden trough padded with straw, but recommends *swaddling* the baby with woollen, soft and clean bandages, three or four fingers' breadth wide, in order to straighten its limbs. The swaddling should be done carefully so as to keep the baby *warm but without using any undue pressure.* All the bony parts are to be protected against compression by means of wool.

Chapter XXIX.—After the bath and swaddling the baby is to be put to *sleep* on a pillow stuffed with wool. He condemns the Thracian and Macedonian method of tying the baby to a hard board. The nursery should be warm, well ventilated, and free from noise or too bright light. The baby is not to be fed for two to three days, since till then it is still filled with nourishment received through the placental circulation. After that time it must be given boiled honey (which Rufus of Ephesus mentions also acts as an aperient). For the first four days after that, Soranus recommends the milk of a woman other than the mother, as this is too thick and cheesy for the baby. He disagrees with Demosthenes, who recommends maternal breast-feeding at once. This custom of abstaining from breast-feeding for two or three days still prevails in certain places at the present day, although it is now well known that the colostrum of which the maternal milk consists during the first couple of days after labour is particularly easily digestible and good for the infant.

Chapter XXX.—In the event of a woman being physically unable to nurse her baby, a *wet nurse* must be employed. The latter must be between 20 and 40 years old; if she is under 20 she is too young, inexperienced and likely to be too careless with the baby, and her milk is likely to be too watery. She must also have given birth to two or three children, since a primipara is not experienced enough and her breasts are too small and too full. On the other hand, if she has had too many children of her own, her milk is equally likely to be too dilute. She must be healthy, as a sick nurse secretes inferior milk. Her breasts must be of proper shape and consistency. If the breasts are too small they contain too little milk; if they are too large the infant cannot drain their contents at each feed and the excess becomes spoilt. The papillæ also must be neither too small to fatigue the baby nor too big to press upon the gums and interfere with deglutition. Moreover, if there are too many ducts the excessive flow of milk might suffocate the baby.

The nurse must be of equable temperament and must live a healthy hygienic life. She must avoid marital relationships, as an intervening pregnancy will spoil her milk. She must also avoid wine which deleteriously affects her milk, giving the baby convulsions. Her own baby ought not to be older than about two or three months, but the sex of her baby is of no importance. Similar directions are given by Paulus Bagellardus (fifteenth century), Bartholomatus Melinger (fifteenth century) and later writers. It will be seen that the rules with regard to the choice of a wet nurse cannot even now be improved upon.

Chapter XXXI describes the **method of testing the characters of the milk.** To tell the quality of the milk from the state of health of the nurse's own baby is, says Soranus, not sufficient, since although a thriving baby indicates a good quality milk, the opposite is not the case, since a baby may not get on even in spite of the milk being good.

To analyse the milk one examines its physical properties. Its *colour* must be moderately white; neither bluish nor greenish, nor reddish, &c. It should have a pleasant *odour*. It should be of uniform *consistence* and contain no fibres, &c. It must be of moderate density or viscosity, since a watery milk is not sufficiently nourishing and too thin a milk is indigestible. The viscosity is tested by putting a drop of the milk on the nail; when the milk is too thin it flows off on the slightest movement of the finger, if it is too thick it moves very sluggishly. Another test is the degree of its *miscibility* with water. The *taste* of good milk is sweet and pleasant. When exposed to the air good milk does not turn sour too quickly and its coagulum is not too thick. The best milk is one which remains good in spite of the nurse's bad diet; the worst milk is one which remains bad even if the nurse's diet is good; medium quality of milk is one which varies with the character of the diet.

The **mode of life of the nurse** is described fully in Chapter XXXII. She must not work hard, nor must she be too idle, because her milk might become too dense and indigestible. Work which entails excessive bending of the body, such as turning a handmill, baking bread, making beds and drawing water, must be avoided. She must keep her bowels well open. After moderate exercise she must massage her breasts with oil, or later in nursing bathe them with warm and cold water alternately (compare Chapter XVI). Her diet must be light, free from condiments, and taken in moderate quantities at a time, since any

derangement of digestion is likely to upset her milk. Soranus enters into great details regarding the particular articles of diet which the nurse should take and those which she should avoid.[1] She is not to drink much water during the first forty days, and if the infant thrives she may have some weak white wine. Excessive amounts of wine may cause convulsions in the infant.

Chapter XXXIII is devoted to a consideration of the methods of improving the quantity and quality of the milk. If the defective quantity or quality is due to any morbid condition of the nurse, such as uterine disease or general malnutrition, then that condition must be remedied. If the milk is too thick, the nurse must drink more water and eat food which creates thirst. She must also have frequent baths. For too dilute a milk the opposite regime must be followed.

In the next chapter Soranus discusses the **bathing and massage** of the baby. Unless the infant is not clean or suffers from skin rashes, no more than one bath a day should be given, and it should not last too long. The bathroom must be suitably warmed and shaded. After the bath the nurse lays the baby on her knees and rubs it in a definite order with warm oil, after which she sponges the whole body with moderately warm water until the skin becomes red. All portions of the body, including the armpits, neck and buttocks, must be thoroughly washed, and the mouth, gums and tongue must be carefully cleaned by means of the finger. Pressure of the hand on the lower part of the abdomen leads to emptying of the bladder. The child must be gradually accustomed to water of lower temperature in order to strengthen its body.

After washing the child is inverted and held by its feet head down in order to straighten its spine and strengthen its muscles. The infant is made to go through a regular system of **gymnastic exercises**, such as touching the left foot with the right hand and vice versa, touching the buttocks with the feet, and the various regions of the spine are massaged and the various muscles kneaded, in order to avoid spinal curvature and other deformities. The head is rotated round the neck as far as possible to increase its mobility and every other joint of the body is suitably exercised. The nose is massaged and the nostrils and ears properly cleaned.

In Chapter XXXV details are given regarding the **technique of breast-feeding**. The breast should not be given immediately

[1] It is now known that diet has very little influence upon the composition of the milk (see Chapter XIV).

after a bath, because in this stimulated condition it is likely to take more than is good for it. For similar reasons a nursing woman should not put the baby to the breast immediately after her own bath, since the stimulus of the bath has made her milk too strong for the baby. The act of suckling is to take place with the woman in a sitting position, with her chest leaning slightly back, so as not to allow too free a flow of milk into the baby's mouth. The breasts should be frequently alternated; this is done to prevent the baby lying too long on one side, which might cause defective development of one or other arm. The nipples are to be washed before each feed. After a feed the baby is carefully laid in its cot. Under no account may the infant sleep in the same bed as the nurse, for fear of overlaying. Too frequent or too long feeding during the day, but especially at night, injures the child. The baby should not be fed immediately before or during its bath. It is dangerous to breast-feed the baby when it is asleep because of the risk of suffocation as the result of compression of its nostrils by the breast. One must not give the baby the breast every time it cries. Moderate and occasional crying is good for the baby, since it thereby exercises its lungs. Excessive crying, however, is injurious, since it hurts the eyes and tends to cause a scrotal hernia (oculos laedit et intestinorum in scrotum descensus causa fit). There are many causes for a baby's crying other than hunger, e.g., an uncomfortable position, pressure of bandages, constipation, irritation of skin by parasites, excessive amount of food in the stomach, cold, &c. Soranus describes the simple differential diagnosis of these causes and advises treatment by removing the cause. Thus crying due to colic is accompanied by eructations aud vomiting, as well as by drawing up of its legs; whilst the cry of hunger is diagnosed by the following signs: (1) absence of any other cause; (2) the movement of its lips and opening of its mouth on touching the same with the fingers, since this reminds the baby of the contact of the nipple with its mouth. Other helps are: (3) the interval since the last feed and the amount of milk then taken by the baby. After feeding, the baby should not be cradled or moved too much, since the movements may have the same effect as the movements of a ship, resulting in sickness. If the baby continues to cry after a feed, one must not lose one's temper with it, since fear excited in a baby may affect its physical and mental development, but one must try to pacify it by kindness, by gentle soothing sounds as well as by means of toys. The baby

must be accustomed to exercise by moving it first in its cradle (scapha), afterwards in perambulators. After the fourth month the nurse takes it out for a walk in her arms.

Chapter XXXVI.—The cord falls off on the third or fourth day Swaddling is to be discontinued between the fortieth and sixtieth day (Chapter XXXVII). It is to be done gradually, and the right arm is to be released before the left so as to avoid left-handedness. (Prius autem dextra exsolvenda; nam vinculis contenta, secundum coram morem, qui sinistram prius solvunt, debilior fit quia tardius quam sinistra ad exercitationem devenit ita ut vel hac de causa nonuli laevi fiant.) When the infant attempts to rise and sit up it is necessary to help it so as to prevent spinal curvature (Chapter XXXVIII). Similarly, when it makes attempts at walking one must see that it does not do so prematurely in order to prevent curvature of the legs. The frequency of curved legs (probably rickets) in Rome and other cities as compared with that in the provinces is due to the fact that *Roman mothers have less time to devote to the care of their babies than have Grecian mothers*. Other people, says Soranus, are of opinion that such curvatures are the result of cold or conceptional parental alcoholism—but he quite rightly does not agree. He recommends the use of go-carts.

Chapter XXXIX.—Weaning must be gradual and neither too early nor too late. No other food in addition to the breast may be given before the sixth month. At that time one may begin with milk, bread soaked in milk, water or honey. But no food may previously be chewed by the nurse. Later one may add a little gruel and eggs. Premature feeding with farinaceous foods has a deleterious effect on the growth and development of the baby. Weaning should begin at the age of about 1½ to 2 years and not in the autumn nor when the baby is ill. Spring is the best time. Female babies should not be weaned later than male infants. No bitter substances should be put on the breast for the purpose of weaning, as these cause intestinal irritation in the infant.

Chapter XL.—The eruption of teeth begins in the seventh month. To allay the irritation of the gums the latter may be rubbed with a finger dipped in oil. In cases of difficult dentition the gums should be incised.

Soranus does not deal with complications of dentition, but Aetius (quoted by Kroner) speaks of pyrexia, aural pruritus and diarrhœa; and Hippocrates mentions convulsions as a complication. At the present day it is believed that dentition will, in

predisposed infants, cause diarrhœa and convulsions, but incision of the gums is very rarely practised.

The subsequent chapters deal with treatment of infantile diseases, with which, however, we are not concerned in this book.

Wet nursing was prevalent amongst the Romans, as it was also among the Egyptians. The rich patricians used to buy slaves to act as foster-mothers even before the baby was born; the poor plebeians hired wet nurses to feed their babies from time to time at a special bureau in the Forum Olitorium near the Colonna Lactaria. This practice became so common that Juvenal satirized it, and Cæsar reprimanded the mothers for paying more attention to their dogs than their babies, whom they entrusted to the care of the "Assae Nutrices" (as these wet nurses were called). Tacitus attributes the lack of men of outstanding ability in Rome to the fact that the mothers did not themselves feed their babies, and thus gives adherence to the unfounded belief that moral and mental traits are transmitted by the milk.

The most remarkable essay against the use of wet nurses was written by Gellius, a Roman lawyer of the second century A.D. In his "Noctes Atticae," a series of discourses on sociology and other things, he records (Book xii, Chap. I) a speech of his teacher and friend, the philosopher Favorinus, in which he urges a noble lady not to hand over her children to wet nurses, but to feed them with her own milk.[1] He points out how unnatural it is for a woman to have given birth to a child and immediately to hand it over to somebody else; to have nourished in her own womb, with her own blood, something which she has never seen, and to refuse to feed with her own milk the object which she now sees imploring the tender care of a mother. "Do you believe," he continues, "that kind nature has given women fullness of bosom and beautiful nipples for the purpose of bodily ornament and not for the purpose of feeding their babies?"

Nursing, he says, does not destroy the mother's beauty. "The natural affection of a child is directed to that object from which it receives its nourishment, and as a consequence the child, having no knowledge of the mother, does not regret her loss." He also speaks of the transference of the physical and moral characters of the foster mother to the baby by means of

[1] Dissertatio Favorini philosophi qua sauvit nobili feminæ, uti liberos quos perisset, non nutricum aliarum sed sibi suo lacte aleret.

the milk.[1] Plutarch ascribes the possession of two breasts by the mother to nature's provision for the need of twins.

Feeding vessels for babies have been discovered in ancient coffins of Greek and Roman infants (figs. 14 and 14A). The food used was, as we have seen, honey, milk, pap and butter (cf. Isaiah vii. 14). The Romans were aware of the great nervous

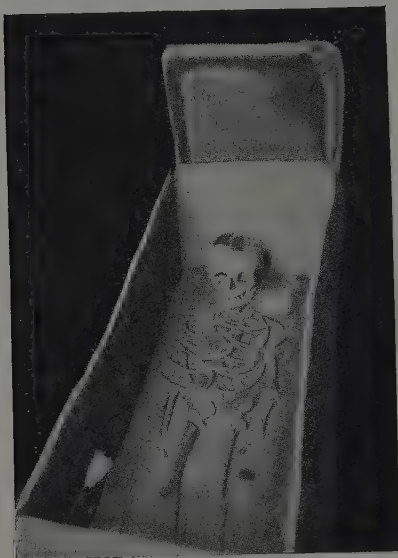


FIG. 14.

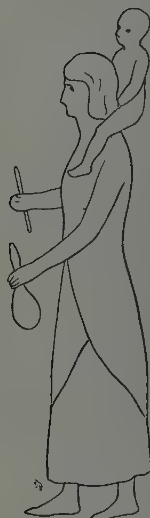


FIG. 14A.

FIG. 14.—Infant's skeleton (with feeding bottle to the left of the skull) found in Aquincum, ancient citadel near the site of Budapest. (From Bokay's "Die Geschichte der Kinderheilkunde.")

FIG. 14A.—Relief of the 9th Century B.C. discovered in the excavations of Nineveh. It shows a mother carrying a baby on her shoulders—as was the custom in Asia Minor—and holding a feeding bottle in her left hand and a rod in her right hand. The latter was probably used for dipping into the bottle and giving the baby to lick. (After H. Pless, "Das kleine kind vom Tragbett bis zum ersten Schritt.")

sensitiveness of puerperal woman, and a sign was placed before her door to keep away visitors—even tax collectors! (De Lee.)

The first laws for the protection of infants were made in Rome. There was a long struggle in ancient Rome against

[1] Early Chinese writers also believed in the transference of moral characters by the milk and refer to "that almost human" intelligence that was acquired by the Imperial Pekingese dogs that were nourished on human milk.

infanticide, and the "patria potestas" which endowed the father with the power of life and death over his children. When a baby was born the midwife placed it at the feet of the father. If he picked it up it became a member of the family; if, however, he turned away from it, it was killed. In the eighth century B.C. Numa Pompilius (as we saw on p. 32) passed a law requiring the removal of the child from the body of a woman who died during pregnancy, in the hope of saving its life, but the patria potestas continued until A.D. 4, when Augustus Cæsar abolished it. Colonies for poor families to enable

FIG. 15.—Tablet in the Forum at Rome commemorating the beneficent deeds of the childless emperor Trajan (who reigned between A.D. 98 and 117), erected in the early years of his reign. On the right it shows Trajan seated in his curule chair (sella curulis) and standing in front of him is a woman with a child in her arms. The woman is generally supposed to represent Italia thanking the emperor for his good act for the maintenance of poor children. Another possible interpretation, given by the late Mr. Benjamin Broadbent, in a private letter to the author, is that the woman has just received back with gratitude from the emperor's hand her child whose life he, as pater patriæ, has just saved by exercising the right of patria potistas.

parents to rear their children were founded in A.D. 97, during the reign of Nerva, and in A.D. 100, in the reign of Trajan, some 5,000 children were cared for by the State. The first institution for the protection of girls and orphans was founded in the second century A.D. by Antoninus Pius in honour of his wife Faustina, who though she was a woman of loose habits had many honours bestowed upon her by her husband. A coin of the period (fig. 16) shows on one side Faustina, and on the other Antoninus surrounded by children and inscribed "Puellae Faustininae." The same

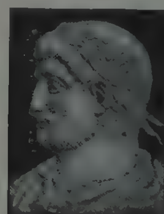
Antoninus rewarded mothers who nursed their babies successfully. Hadrian and Marcus Aurelius, as well as Pliny, made similar benefactions.

FIG. 16.—Coin showing Puellae Faustinae.

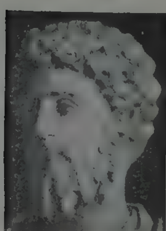
(*Reproduced, by permission, from a gold coin in the British Museum.*

Augustus Cæsar.

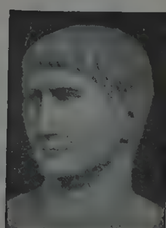
Nerva.



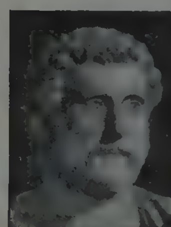
Constantine.



Marcus Aurelius.



Trajan.



Antoninus Pius.

FIG. 17.—Pagan emperors of Rome (with the exception of Constantine), who legislated for the protection of children.

According to Cicero, Camillus, when Consul, proposed a tax on bachelors for supporting infants (Holt).

Constantine, in the early years of the fourth century, in order

to discourage the abandonment of infants from economic causes, offered money to poor parents to supply the needs of their new-born infants. He made infanticide a capital offence in 318 A.D., and decreed the same punishment for it as for parricide, viz., "That the murderer be sewn up in a sack with a dog, a cock, a viper and a monkey, and thrown into a river or the sea." In the latter part of the same century, the Emperors Valeus and Gratian ordered that every parent should bring up his own infants, and they pronounced penalty against exposing them. Julius Paulus, a Stoic in the reign of Severus, considered induction of abortion, as well as exposure of infants, akin to murder, but owing to the great poverty of the Roman Empire the Stoics could do nothing to stop these evils.

The **Byzantine period** (A.D. 476-732), or the period of the East Roman Empire (embracing Egypt, Thrace, Macedonia and Greece), may be omitted from our survey, since there is little in the writings of that period which is relevant to our subject. But **the year 787** is a red-letter date in the history of the infant welfare movement, since in that year **Archbishop Datheus**, of Milan, founded the first asylum for abandoned infants. In his edict Datheus expresses his wish that, "as soon as a child is exposed at the door of a church that it will be received in the hospital and confided to the care of those who will look after it. These infants will be taught a trade, and my wish is that when they arrive at the age of 8 years they will be free from the shackles of slavery and free to come and go wherever they will." Between the eleventh and fifteenth centuries many foundling hospitals were established in different parts of the world. Pope Innocent III, at the end of the twelfth century, arranged that unwanted babies should be admitted into the Hospital of the Holy Ghost in a manner which ensured failure of identifying the mothers. The mother placed the baby in a "turning box," which was working in a pivot in the wall of the hospital, and then rang the bell, when the box was turned round on its pivot by an attendant from inside the hospital. This system gave the hospital the name of Conservatory of the Wheel (Conservatorio della Ruota). This system of strict secrecy, aided by the fact that the infant was thereafter considered legitimate, replaced the evil of legalised infanticide by that of legalised abandonment. In 1523 the Hôtel Dieu of Lyons, the oldest hospital in France, began to take in children. A prominent pioneer in the seventeenth century was **St. Vincent de Paul**

(1576-1660), who in 1639 put on a firm foundation the asylum for exposed infants started by an unknown woman of Paris. The asylum was officially recognised by Louis XIV in the year 1670.

It is related of St. Vincent, who was originally a peasant, that he used to roam about the streets of Paris in search of deserted babies whom he carried to his asylum. Once he saw a beggar ill-treating an abandoned baby, when he rushed up to him and exclaimed, "Barbarian, how deceived I was; I thought, from a distance, you were a man," and snatching the baby ran with it through the streets of Paris, calling upon everybody to follow him. On reaching the asylum he pleaded to his followers for support for his institution. St. Vincent was also the founder of the Order of Lazarite and of the Sisters of Mercy.

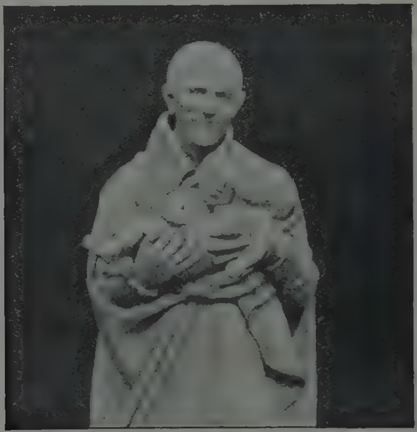


FIG. 8.—St. Vincent de Paul.

The two foundling hospitals in Russia (in Moscow and St. Petersburg) were founded in 1763 and 1770 respectively by the order of Empress Katherine the Great.

In the **fifteenth and sixteenth centuries** artificial feeding of infants was very prevalent, milk of the cow or goat, or bread and sugar, being used in the absence of breast-feeding. The date of weaning was generally determined by the appearance of dentition, until it was realised that this was an inconstant factor and that some infants are born with teeth, e.g., Louis XIV of France and Richard III of England, of whom Shakespeare makes the young Duke of York say:—

> "Marry, they say Uncle grew so fast
> That he could gnaw a crust at two hours old."
> RICHARD III, Act ii, Scene 4.

Several poems dealing with the hygiene of infancy written by doctors and others, also appeared during the same period. Heinrich von Louffenburg (a German monk), in 1429, wrote "Versehung des Leibs," containing a portion dealing with the hygiene of the expectant mother and the infant. Luigi Tansillo (Italian poet and soldier, 1510-1589) wrote a poem called "La Balia," which was translated by William Roscoe in 1798. Claude Quillet, French physician, poet and clergyman (born 1602), wrote "Callipaedia" ("The Way to have Handsome Children") (see p. 46).

Lastly, Scévole de Sainte-Marthe, a French physician (born 1526), published his "Paedotrophia" ("The Art of Bringing up Children") in 1584. He refers to dentition as the time for weaning:—

> "Since with the Breast he must long be fed,
> His Growing Teeth prepares his Age for Bread,
> For when eight Moons have run their wonted Race
> The Fluid to the Solid Meal gives place."

Books dealing with pædiatrics, and incidentally with child hygiene, in the fifteenth and sixteenth centuries are those by Paulus Bargellardi (Padua, 1472), Bartholomaeus Metlinger (Augsburg, 1473) and Cornelius Roelanus (Mechlin, 1483). The second of these gives a very complete account of the hygiene of infancy in many respects resembling the treatise of Soranus. In the sixteenth century appeared the works of Leonellus Faventinus de Victoriis (1544), of Hieronymus Mercuriale (Bologna, 1513), of **Thomas Phaer**, the father of English Pædiatrics ("The Boke of Children," 1544, the first of its kind in English), of Sebastinus Austrius (Basle, 1549), and works in Spanish, of which the most important is that of Jeronimo Sorane, who advocates starvation treatment for cases of infantile diarrhœa. The first book on infant hygiene in France was that of Simon de Vallembert, Physician to Marguerite of France, in 1565.

Till that time pædiatric knowledge remained at the same level at which it had reached in the times of Celsius, Soranus and Galen. Swaddling was still practised, and a painting of the period represents Ludwig XIV as a new-born baby fully swaddled, but for its arms which are left free.

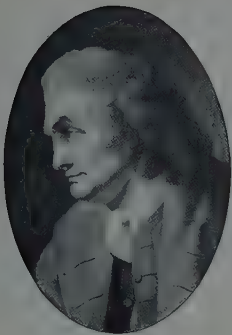
Scarification of the gums to help eruption of teeth, a practice recommended by Soranus only in cases of difficult dentition, was widely practised during the three hundred years between the sixteenth and nineteenth centuries. This was introduced by the famous French surgeon, Ambroise Paré, in the sixteenth century, as a new method destined to cure all the troubles that were believed to be associated with dentition. It was persisted in with such enthusiasm that John Hunter, in 1772, actually performed this operation twenty times in one little patient until the tooth ultimately appeared! (Kassowitz, quoted by Bokay). Paré also interested himself in the question of infantile syphilis, and the following observation made by him is of interest. A wet nurse was in one case employed to supplement maternal breast-feeding. The nurse happened to be syphilitic and infected the baby, who gave the disease to its mother, from whom in turn its father was infected, and the latter ultimately infected two of his other children.

In 1620 the microscope was invented, and in 1638 the thermometer was introduced. Both these played important parts in the subsequent development of child hygiene on scientific lines. In 1667 Swammerdam showed that the lungs of a new-born infant that had breathed floated in water. This test was used medico-legally for the first time in a case of infanticide in 1687, by John Schreyer. A work of considerable historical interest is that of a Jewish physician, **Dr. Tobias Katz,** rabbi and professor of medicine in Constantinople. It is written in Hebrew ("Maase Tuvia," i.e., the work of Tobias) and was published in 1695. It contains some interesting matter regarding the ante-natal, intra-natal and post-natal welfare of the child.

There were several pioneers in the period we are now considering. **Francis Glisson,** of Dorsetshire (1597-1677), published his classical work on rickets, "De rachitide, sive morbo puerili qui vulgo the rickets dicitur tractatus,' in 1650 [1] (second edition, 1660). This disease which, as we have seen, was recognized by Soranus in the second century A.D., was (according to Garrison) first mentioned in the English Bills of Mortality in 1630. Claude Quillet, a physician of the seventeenth century, writing under the pseudonym of "Calvidius Letus," published a poem at Leyden, in 1665, named "Callipaedia seu de Pulchrae Prolis Habendae Ratione" ("The Way to have Handsome Children"), of which an

[1] Translated into English by Phil Armin, 1651.

MEDIÆVAL PEDIATRICIANS.



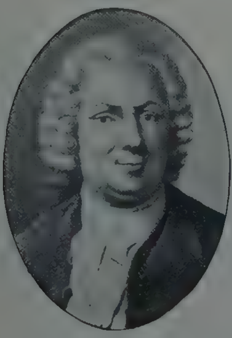
WILLIAM CADOGAN.

FRANCIS GLISSON.

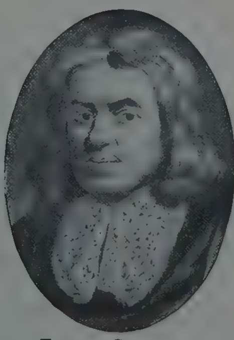
EDWARD JENNER.

TOBIAS KATZ.

GIOVANNI MORGAGNI.



ROSÉN VON ROSENSTEIN.



THOMAS SYDENHAM.

CHILD SOCIOLOGY.

T. J. BARNARDO.

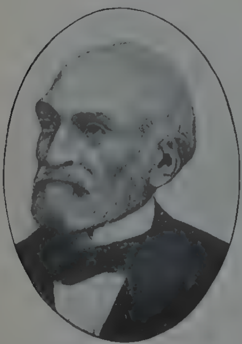
A. EPSTEIN.

E. CHADWICK.

A. JACOBI.

JANET LANE-CLAYPON.

RAUCHFUSS.



TH. ROUSSEL.

J. CRICHTON-BROWNE.

CHARLES WEST.

English translation was published in London in 1710. In this poem he deals with matters of hygienic importance in a thoroughly practical manner:—

> "You now, who are disposed to learn our Arts,
> Imprint this useful Lesson on your Hearts.
> Not all of either sex by Hymen joined,
> Are always apt, or should encrease their Kind.
> Ne'er, when the Body is defil'd, presume
> Within the Temple of God to come.
> . . . .
> . . . . , .
> For if the Generative Seed's defil'd
> The Father's Hurt's transmitted to the Child."

William Cadogan, of London (1711-1797), wrote an "Essay on the Nursing and Management of Children" in 1750. **George Armstrong**, in 1769, established the first Dispensary for Poor Children in London. It was the first of its kind in Europe, but owing to lack of funds was closed in 1781. **Michael Underwood,** of London (1736-1820) recognised that the health and welfare of the adult population depended upon the amount of care and attention devoted to infants, and in 1774 appeared his "Treatise on the Diseases of Children." In this book, which went through many editions, Underwood recognised the importance of preventive medicine. He says: "Whatever splendour the actual treatment of disease may reflect on the science of medicine, it by no means comprehends the whole of its province; for prevention being in every case preferable to remedies, the medical art would be more imperfect than other sciences were it devoted only to the latter." The author also approves of the proposed family endowment. "A small premium given every year to each poor family for every child they have alive at the year's end, would save more infants' lives than if the whole revenue of the Crown were expended on hospitals for that purpose." He attributes the high child mortality in London at that time (viz., "almost two-thirds of the children born in London and its environs die before they are 5 years old, and more than three-quarters of these die before the end of the second year") to want of air and exercise and to improper diet. He speaks of breast-milk as the only natural food for an infant, and says that in Greenland and among the Esquimaux, "whenever a suckling-mother happens to die her infant is buried with her. . . . It is esteemed an act of compassion to put an end to an infants'

suffering (as the result of artificial feeding) by plunging it into the sea."

Giovanni Battista Morgagni (1682-1711), the founder of pathological anatomy, made pathological post-mortem examinations on children, and **Nil Rosén von Rosenstein** (1706-73), a pupil of the famous Dutch physician, Boerhaave, was the Swedish pioneer in the art of pædiatrics. He wrote on diseases of children in 1764, in which he dealt with the various problems relating to infant hygiene. He devotes eighty pages to small-

Fig. 19.—Babies being suckled by asses.

pox, and deals with inoculation against the disease, a method introduced from Constantinople by Lady Mary Wortley Montagu. In the case of ailing nurslings, including congenital syphilis, he administered medicines to the mother or wet nurse in the belief that these would be transmitted to the infant through the milk. When a syphilitic baby was artificially fed, he made it suck from the udders of a goat, and later of an ass, which had been previously shorn and treated by mercurial inunction. The infantile mortality was reduced by this means from

83 per cent. among bottle-fed to 67 per cent. among those fed direct from the goat, and 21 per cent. among those nursed by the ass.

The same was practised by Parrot in the Hospice des Enfants assistés in Paris (fig. 19), with a marked reduction in the mortality of such infants (1880).

The goat as a wet nurse, introduced in the eighteenth century, had its analogue in ancient Greek mythology, where Æsculapius and Jupiter are said to have been suckled by a goat and Remus

FIG. 20.—The statue (in the Capitol in Rome) of the wolf with Romulus and Remus.

and Romulus by a she-wolf, from whose milk they acquired their great physical strength[1] (fig. 20).

The practice was widely used in France, and, as the result of the advocacy of Zwierlein, a German village doctor, was introduced in the early part of the nineteenth century into Germany. The practice was discontinued in Germany about the middle of

[1] Even at the present day accounts not fully substantiated are published from time to time of Indian infant girls, who were exposed by their parents, being found and nursed by wolves, as well as tigresses. On October 22, 1926, the *Westminster Gazette* published a story, said to be vouched for by the Rev. J. A. L. Singh, of Midnapur, Bengal, of two girls, aged 2 and 8 years respectively, who had been found in a wolf's den. "The girls were very fierce. They darted away on all fours . . . uttering guttural sounds." An account of a "tiger child" is given in the *Morning Post* of December 31, 1926.

the nineteenth century, and, after Parrot's death, in France, chiefly on account of the great expense, viz., 2,000 francs a year per baby.

The practice is fairly widespread at the present day in certain Southern States of America, as well as in certain parts of India. Such a method, of course, eliminates the danger to the infant incidental to dirty handling of milk and to its contamination by flies.

The Foundling Hospital of London was founded in 1737 by Thomas Coram.

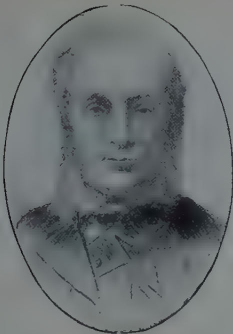
Edward Jenner introduced vaccination against small-pox in 1796, but the lowering of infant mortality brought about by this operation was wiped out by the great industrial changes that took place at that time. The vast improvement in machinery created a demand for cheap labour, which brought thousands of women into the workshops. This caused the migration of women from rural districts into the cities, with the following deplorable results: (1) The mother being out at work had to neglect her children; (2) the increased cost of living in the cities exposed the women to the temptations of prostitution with the consequent increase of venereal disease, which, of course, had a deleterious effect upon the life of the child at every stage of its existence.

In 1761, Jean Jacques Rousseau published "Emile," in the introduction to which he deals with the hygiene of infancy, and the danger of mercenary wet nursing. He exhorts mothers to breast-feed their babies. About the same time, Ballexserd, of Geneva, wrote a "Traité de Puériculture," which gained him a prize from the Haarlem Académie. The author deals with the feeding, clothing and exercises of babies, and recognises the need not only of ante-natal but of ante-conceptional hygiene.

Richard Russell published "A Dissertation on the Use of Sea-Water in Diseases of the Glands" in 1750. He was the first to discover the curative powers of the sea in cases of tuberculous bones, joints, &c., and in 1796 the Royal Sea-Bathing Hospital of Margate was established. Similar hospitals were established later in other parts of the world.

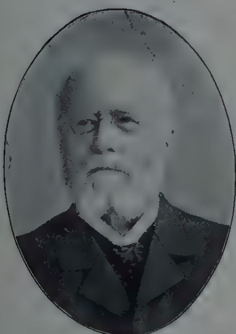
Infantile mortality was very high in the seventeenth and eighteenth centuries, but the mortality amongst breast-fed babies was only one-third that of infants artificially fed. The mortality was particularly high in the foundling hospitals. Thus, in the Dublin Foundling Hospital only forty-five infants are said to

COMMUNICABLE DISEASES.



Bouchut.

Bretonneau.



Klebs.

Trousseau.

Behring.

O'Dwyer.

B. Schick.

COMMUNICABLE DISEASES

George Dick.

Gladys Dick.

Grancher.

Nathan Raw.

Calmette.

have survived out of 10,272 admitted during the twenty-one years, 1775-96, giving a mortality of 99·6 per cent. The chief cause (among others) was lack of breast-feeding. When this lack was supplied in the British Lying-in Hospital the mortality at once fell to 60 per cent. of that in the preceding years. Other causes of high infantile mortality outside the hospitals were baby farming and mercenary wet nursing. The prospect of earning money as wet nurses prompted many young women to have illegitimate children whom they abandoned or otherwise neglected.

The first systematic treatise on *School Hygiene* was written by Johann Peter Frank (1745-1821). His book, entitled "Complete System of Medical Polity" (1777-88), contains practically all the matter in connection with school hygiene, such as lighting, heating and ventilation of schoolrooms, &c., which was later brought out by Virchow and Hermann Cohn. School lunches for children were started by Count Rumford in Munich in 1792.

Nineteenth Century.—The sanitary conditions of hospitals towards the end of the eighteenth century were deplorable in the extreme. In the Hôtel Dieu, in Paris, 100 patients were accommodated in one ward, and several patients, including eight or nine children, were put into one bed at a time, whilst no attempt was made to keep the infectious cases separate from the non-infectious ones. As the result of a commission, consisting of famous physicians and scientists, including Laplace and Lavoisier, which reported in 1785, the **Hôpital des Enfants malades**, the first children's hospital in Europe, was founded in Paris in 1802, under the directorship of Jadelot. Eight years after, Lavoisier met his death at the guillotine.

The establishment of this hospital marks the beginning of the scientific study of the diseases peculiar to children, and in virtue of the association with it of such eminent physicians as Trousseau (1845-53), Roger (1853-74), Bouchut (1863-83), and others, it became a great teaching centre for students of pædiatrics all over the world, and made France the foremost country in the world for the scientific study of the hygiene and pathology of child life. The second children's hospital was established in Russia in 1834, by Friedeblitz. These were followed by children's hospitals in Austria (founded by K. W. Mauthner), Germany, and other parts of the world. The Hospital for Sick Children, Great Ormond Street, London, was founded in 1852

by **Dr. Charles West.** In 1817 John Bunnell Davis published his classical book on "Child Mortality," and it was he who instituted health visiting amongst the poor with the object of giving instruction to ignorant mothers in the art of caring for their infants. In 1826 Bretonneau first described diphtheria, and in 1813 his pupil Trousseau first introduced tracheotomy (an operation mentioned in the Talmud, in the case of animals suffering from wounds of the neck, though not recorded as practised in human beings) into the treatment of croup. The Registration of Births and Deaths became compulsory in England in 1837. In the same year the French physician Colles (together with Baumes) described the fact known as **Colles' law**, that whilst a congenitally syphilitic baby will infect a foster-mother, it never infects its own apparently healthy mother. The comparative chemical compositions of human and cow's milk were first extensively investigated in 1838 by Johann Franz Simon, of Berlin. In 1840 the first English Vaccination Act was passed. In 1842 gymnastic exercises became obligatory in German schools, and medical supervision of schools was introduced into France. The first pædiatric journal, the *Journal für Kinderheilkunde*, was founded in Berlin, in 1843, by Barez and Romberg. In the same year Andral and Gavaret did a couple of gaseous metabolism experiments in children. In 1844 **M. Firmin Marbeau,** Mayor of the First Arrondissement of Paris, founded the first crêche in the world. The idea soon spread all over Europe and other parts of the world. In England, Robert Owen, as long ago as 1800, opened an infants' school which was in certain respects the prototype of the modern crêche. Up to that time women and children (even under 9 years of age) worked in factories during the night, often under conditions of excessive heat and pestiferous stench. Many young expectant mothers were obliged to stand for twelve hours a day. In 1847 a ten-hours-a-day Bill was passed, and children under 9 years were not allowed to work in factories. In 1855, the *Oesterreichisches Jahrbuch für Padiatrik* was published by B. Kraus and L. Mauthner. This ran for three years only. The *Jahrbuch für Kinderheilkunde* was established in 1858, as the result of the activity of Franz Mayr, who also discovered, in 1852, the fact that measles is transferable by means of nasal secretions and tears. In 1858 also, Emil Bouchut suggested intubation for laryngeal obstruction, but gave up the idea because it was not approved by the French Academy of Medicine. It was only when O'Dwyer, of New York, in 1887,

first carried out the operation practically that it was taken up by Hutinel, in France, Bokay, in Hungary, and by others. In 1854 an anonymous writer, "Philopedos" (lover of children), was instrumental in establishing the first children's hospital and nursery in New York. In the same year the Clinical Hospital and Dispensary for Children was founded in Manchester by Whitehead and the Hungarian immigrant Schöpf-Merey. In 1867 Rauchfuss established, in St. Petersburg, what was for a long time the most modern and largest children's hospital in the world. He was also responsible for the plans of the St. Vladimir Children's Hospital in Moscow. In 1862 Petenkofer published the results of his investigations on the effect of defective school ventilation upon the health of school children, and a few years later, Hermann Ludwig Cohen (1866-67) and Rudolf Virchow (1869) wrote on defective sight and various other aspects of school hygiene. In 1865 Dr. William Farr dealt with "Infant Mortality" in his Registrar-General Reports for England and Wales. In 1868 the *Jahrbuch für Kinderheilkunde* contained an article, by A. Steffen, advocating the compulsory education of medical students in the physiology and pathology of child life, and in 1867 Allix published in Paris his *Etude sur la physiologie de la première enfance.* In 1869, Philip Biedert put forth the theory that the indigestion of artificially-fed babies was caused by the casein of the cow's milk. In 1869 Soxhlet introduced the idea of surgical cleanliness into the hygiene of cowsheds and of milk transportation. He also introduced milk sterilization (1886), about the value of which, however, opinions are not now in agreement. In 1870 Ritter v. Rittershain and Maximilian Herz published the *Oesterreichisches Jahrbuch für Kinderheilkunde*, which ran only for three years. Sir Edwin Chadwick was the principal author of a Report of a Royal Commission, in 1833, on the investigation of the conditions under which children were working in factories, which was responsible for the limitation of their hours of work to six daily and for the establishment of the half-time system of education for factory children. He also wrote on schools as centres of school epidemics and the sanitary principles of school construction, in 1871. Baginsky published his "Handbook of School Hygiene" in 1879. In 1872 the Life Protection Act was passed, requiring registration and inspection of all places where infants were farmed out. A similar Act, the loi Roussel (after the famous French doctor,

statesman and sociologist, Théophile Roussel) was passed in France in 1874. These laws were followed by legislation against the employment of women in factories for a statutory period before and after confinement. The first country to pass such an Act was Switzerland, in 1877, when it was enacted that women must have eight weeks' rest, viz., two weeks before and six weeks after confinement. In 1874 the Births and Deaths Registration Act was passed, which regulated the registration of births compulsory since 1837.

In 1867 Dr. Thomas John Barnardo opened the first Dr. Barnardo Home for Waifs and Strays, in Stepney Causeway, for the housing, feeding, education and industrial training of destitute children. By the time of Barnardo's death in 1905 there were 112 district "Homes" in connection with the main institution, and some 60,000 destitute and homeless children were rescued.

In 1875 **Klebs** gave the first account of the diphtheria bacillus, which was first grown in culture by Löffler, in 1884. The organism is therefore known as the Klebs-Löffler bacillus. The discovery of diphtheria antitoxin was made by **Behring** and Kitasato in 1890, and its practical use was shown by Roux at the Hygiene Congress at Budapesth, in 1894. Washbourn, Carel and Goodall, of the Homerton Fever Hospital, London, published their first results with the antitoxin before the Clinical Society of London in December, 1894, when they showed a reduction in diphtheria mortality by 50 per cent., and a reduction in the fatality of tracheotomy cases from 71 per cent. to 47 per cent. In 1913 **Bela Schick,** of Vienna, introduced the Schick test for immunity to diphtheria, and the use of toxin-antitoxin for immunization purposes against the disease, suggested by Theobald Smith in 1907, was clinically applied for the first time by Behring in 1913. The great Public Health Act was passed in 1875. The *Société d'Allaitement Maternelle* was founded in Paris in 1876; this encourages breast-feeding by mothers. The *Central Zeitung für Kinderheilkunde,* established by Babinsky in 1877, became the *Archiv für Kinderheilkunde* in 1879. In 1877 Joseph Forster was the first to study the gaseous metabolism of new-born babies, and he showed that they produced twice as much CO_2 per unit body weight as adults. These investigations have been subsequently continued by Richet, Langlois, Mensi (1894), Scheer (1896), Babak (1901), Hasselbach (1904), Weiss (1908), Carpenter and Murlin (1911), Birk and Edelstein (1910), and others, but in 1898 Rubner and

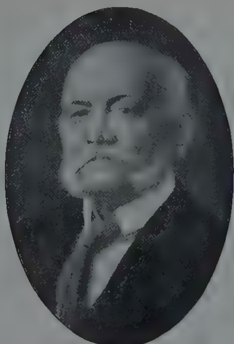
NUTRITION.

P. BIEDERT.

E. MELLANBY.

A. CZERNY.

TH. ESCHERICH.



FRANZ V. SOXHLET.

H, FINKELSTEIN.

CL. PIRQUET.

T. M. ROTCH.

E, HOLT.

INFANT METABOLISM.


O. HEUBNER.


M. RUBNER.


G. VARIOT.


A. B. MARFAN.


FRITZ TALBOT.


A. SCHLÖSSMANN.

Heubner published their brilliant researches on the food requirements of normal and wasting babies, which showed that metabolism in human beings, as in animals, is a function of the surface area of the body (*law of surface area*). This is generally known as *Rubner's law*, although in France it is referred to as Richet's law. Numerous other workers have since then carried out similar experiments, e.g., Variot in conjunction with St. Albin (1903), and with Lavialle (1912), but most of all **Talbot,** together with Benedict and others of America, have, from 1914 onwards, done an enormous amount of exact experimental work on the subject of metabolism in children from birth onwards. Their results do not agree with those of Rubner and Heubner (see Chapter X).

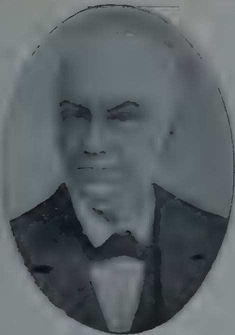
In 1878 Friedrich Ahlfeld, of Leipzig, first introduced the idea of studying the progress of babies by weighing them. In 1880 Tarnier and Auvard introduced incubators into the Paris Maternité, and thus reduced the neo-natal mortality of premature infants under 2 kg. ($4\frac{1}{2}$ lb.) from 66 per cent. to 38 per cent. The value of incubators is now being disputed, and the present writer has succeeded in rearing a premature baby, about $1\frac{1}{2}$ lb. in weight and 13 in. in length, without the aid of such an apparatus. In 1880 **Abraham Jacobi** founded the pædiatric section of the American Medical Association, and in 1888 the American Pædiatric Society. In 1881 the *Revue Mensuelle des Maladies de l'Enfance* was issued. In 1882 school lunches became established by law in France. The *Revista de enfermedades de Ninos* was started in Madrid in 1883. In the same year Thomas Barlow published his classical paper on infantile scurvy. In 1844, Credé introduced silver nitrate instillation into the eyes of new-born babies for the purpose of preventing ophthalmia neonatorum, a mode of treatment which, as we have seen, had to a certain extent been practised by Soranus in the second century (see p. 33). In 1884 Dr. (now Sir) James Crichton-Browne published a report to the Education Department upon alleged over-pressure of work in public elementary schools. W. Preyer's book, *Specielle Physiologie des Embryos*, appeared in 1885. Intubation of the larynx was announced by J. O'Dwyer, of the Foundling Asylum of New York, in 1887. In 1888 Ritter v. Rittershain, director of the Prague Foundling Asylum, founded the *Jahrbuch für Physiologie und Pathologie des ersten Kindesalters*, which ran for a few years only. In 1889 was started *Archivo italiana di pediatria*, and was continued for five years. The Infectious Disease Notification

Act in England was passed in 1889, and in 1890 the *Société de Pediatrie* was founded in Paris. The *Archives de Médecine des Enfants* was first issued in 1897, and a new Vaccination Act, making the use of calf lymph compulsory (instead of arm-to-arm vaccination), as well as allowing conscientious objection, was passed in 1898. In 1890, Professor Hergott, of Nancy, opened the first "Infant Consultations" in France, but the first fully developed institution of the kind was opened by Dr. **Pierre Budin,** in Paris, in 1892. Such institutions, whose object is to encourage breast-feeding, rapidly multiplied in France and in other countries, as follows: Belgium 1897, Italy 1900, Switzerland 1901, Hungary 1902, Spain 1904, Germany 1905, and England 1906. Milk stations, subsequently called *gouttes de lait*, which distributed clean milk to necessitous mothers who could not breast-feed their babies, were first established in Paris by Variot in 1892. The following year similar stations were founded in New York by Nathan Straus, and a depot of the same kind was first opened in England in St. Helens in 1899. The first *goutte de lait* in Russia was established in Kiev, in 1893, by Prof. Troitzky, and infant consultations were first started there in 1906. At first the *gouttes de lait* were accused of encouraging artificial feeding, but very soon they became complementary institutions to the infant consultations. Dr. Henri de Rothschild (1872) has done a very great deal of valuable work in infant welfare in Paris, and Henry Ashby (1845-1908) did similar good work in Manchester. Von Behring, as stated above, introduced antidiphtheritic serum, which has so profoundly affected the mortality rate of diphtheria, in 1890. Its first practical results were shown by Roux in 1894. In 1898 Henry Koplik discovered the spots diagnostic of measles in the prodromal stage (Koplik spots).

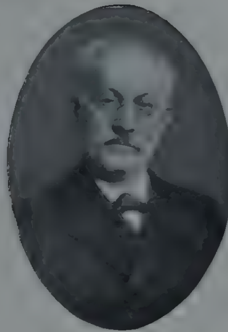
Twentieth Century.—At the beginning of the present century infantile mortality (i.e., the mortality of babies under one year old) continued at practically the same high level of about 150 per 1,000 live births, as in the middle of the last century; and this in spite of the gradual decline in the general mortality brought about by the sanitary improvements resulting from the Public Health Acts of 1872 and 1875. It was therefore realized that there must be some factors in addition to general sanitary improvements which have an influence on infantile mortality. One of these specific factors was found to be some derangement of digestion due to improper feeding, for it was found that the

PLATE VII.

EDITORIAL AND PUBLICITY.



STEFFEN.

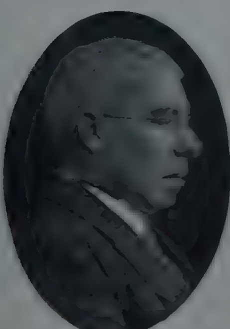


A. BAGINSKY.

GEORGE CARPENTER.

LUIGI CONCETTI.

J. D. ROLLESTON.

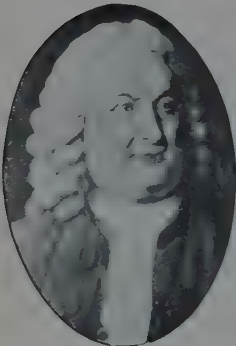


T. N. KELYNACK.

C. W. SALEEBY.

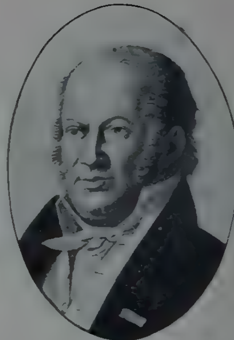
PLATE VIII.

ANTE-NATAL LIFE.

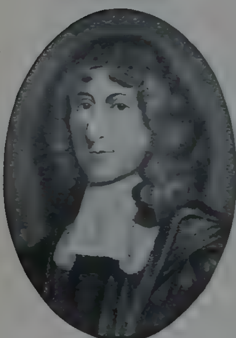


VON HALLER.

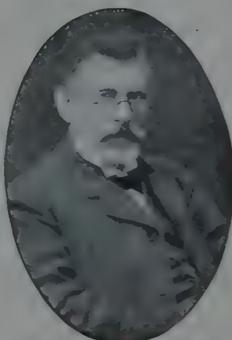
A. WASSERMANN.



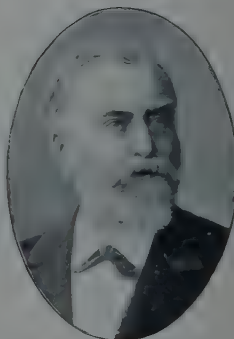
G. ST. HILAIRE.



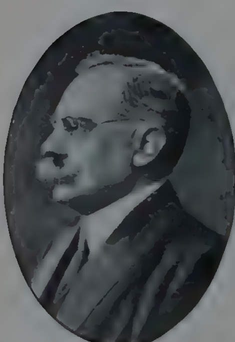
JOHN MAYOW.



P. EHRLICH.

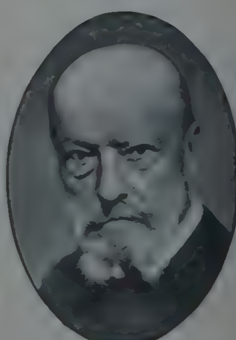


PAUL ZWEIFEL.



JOHN WILLIAM BALLANTYNE.

F. J. BROWNE.



A. PINARD.

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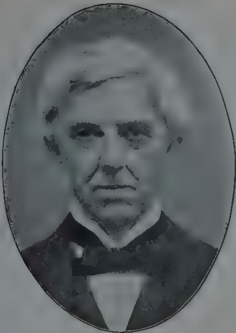
mortality amongst babies artificially fed was much higher than that of babies fed on the breast. The campaign for the reduction of infant mortality concentrated itself, therefore, upon two main principles, viz.: (1) encouragement of breast-feeding; (2) perfection of the methods of artificial feeding. Hence, the study of the physiology and pathology of early life—especially research in the subject of infantile metabolism—which was seriously begun in the nineteenth century, has, during the twenty-five years of the present century, been pursued with accelerated speed and with intensified vigour throughout the civilized world. In addition, much philanthropic and sociological work has been done in connection with the subject of child welfare. All these efforts, as we shall see in Chapter V, were instrumental in bringing down the infantile mortality from about 150 to the comparatively very low, but absolutely still high, figure of about 70 per 1,000. In this century also it has been realized that, in order further to reduce infantile mortality, two other factors must be considered in connection with the subject of infant welfare, viz., neo-natal care, or care of the infant during its first month of life, the period of life during which the mortality has remained almost stationary, and ante-natal care, or care of the baby before it is born.

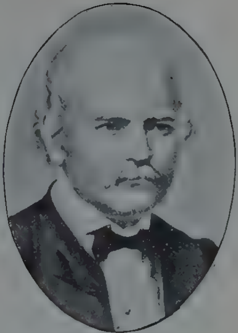
John William Ballantyne, of Edinburgh, was the first in the world to have brought the subject of ante-natal life prominently to the fore, and the two volumes of his monumental work on ante-natal pathology and hygiene, which laid the foundation of the scientific study of ante-natal life from the point of view of preventive medicine, appeared in 1902 and 1904. His indefatigable advocacy succeeded in persuading the authorities of the Royal Maternity Hospital, Edinburgh, to set aside one *prematernity bed* in 1901 for the purpose of the treatment and the scientific investigation of the abnormalities of pregnancy. This single bed grew and multiplied until at the time of his death in 1923, the first prematernity centre in Europe was established in Edinburgh containing no less than twenty beds! In addition, the ante-natal clinic which he established has been the starting point of numerous similar clinics throughout the world. In England alone there are now about 600 ante-natal clinics in connection with infant welfare centres, or attached to maternity institutions. The French protagonist in the subject of ante-natal hygiene is **A. Pinard.** In 1900 **J. Grancher** introduced the "**Box System**" into the Paris Hôpital des Enfants malades, and was thus able to reduce the measles mortality in the hospital

from 25 per cent. to 5 or 6 per cent. This system is now adopted in many hospitals in France, including the Pasteur Hospital for Infectious Diseases. In 1902 school lunches were introduced in England, and dental clinics were started in German schools. In the same year Dr. Robert Hutchison delivered the first course of lectures on "Diseases of Children," at the London Hospital Medical College. The Midwives Act, which did away with unqualified and untrained midwives, was passed in 1902 and came into force in 1905. Women who had been in bona-fide practice as midwives before then were put on the register and were given special theoretical and practical instruction in midwifery and neo-natal hygiene under the auspices of the various municipal bodies. The present writer was one of the lecturers under that scheme for the London County Council. About the same time, also, the London County Council introduced free lectures on Infant Care into their Evening Classes as well as, on request, into Girls' Clubs and Mothers' Meetings, and the author was one of the early lecturers under that scheme. In 1903 Mr. Robert Mond founded, in memory of his wife, the Infants Hospital, London, for the purpose of treating and investigating diseases of nutrition in infants. Dr. Ralph Vincent was its senior physician from the time of its foundation until his death in 1922. Its present medical director is Dr. Eric Pritchard, who has associated himself with infant welfare organisations in London. In 1903 also was founded the *Rivista di clinica pediatrica* (edited by Comba and others). The *British Journal of Children's Diseases* was founded in London by George Carpenter in 1904. Its present editor is Dr. J. D. Rolleston, who has written extensively on infectious diseases in children. In 1906-9 Czerny and Keller disputed Biedert's theory of casein indigestion and replaced it by the theory of fat indigestion. The first National Conference on the Infantile Mortality was held in London in 1906, and in the same year the first infant consultation was started by Eric Pritchard at Marylebone, London. In 1907 the first school for mothers was started by a number of ladies in St. Pancras, London. Dr. Janet Lane-Claypon, who, together with Professor Starling, showed experimentally, in 1906, that lactation is independent of the integrity of the nerves supplying the mammary glands, studied the methods of prevention of infant mortality in various European countries in 1910. She also wrote extensively on milk and the child welfare movement.

MATERNAL MORTALITY.



OLIVER WENDELL HOLMES.



I. SEMMELWEISS.

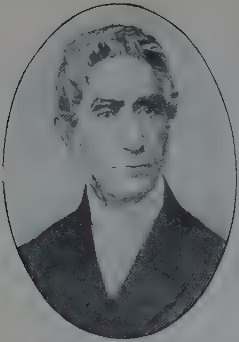
JANET CAMPBELL.

PASTEUR.

LISTER.

p 58

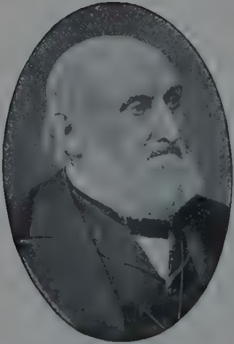
ANTHROPOMETRY.



H. QUETELET.

F. LIHARZIK.

MARIA MONTESSORI.



W. FARR.

A NEWSHOLME.

p 59

Owing to the great success of the notification of births within thirty-six hours (instead of the ordinary registration within six weeks), which was made compulsory in Huddersfield in 1906 as the result of the advocacy and activity of its Medical Officer of Health, D. S. G. Moore, and its then Mayor, Benjamin Broadbent, the Notification of Births Act was passed as an adoptive Act for the whole of England in 1907, and was made a compulsory measure in 1915. This Act made possible the immediate skilled supervision of new-born infants. Broadbent and Moore's work was inspired by the good results obtained by M. Morel de Villiers, Mayor of the village Villiers-le-Duc, in France (where an average infant mortality of 154 per 1,000 in 1884-93 was altogether wiped out during 1893-1903), and was responsible for the lowering of the infantile mortality in Huddersfield from 134 in 1906 to 97 per 1,000 in 1907.[1] In 1907 the present writer published the first book of its kind in Yiddish on the subject of "Infant Care" (Das Kind). This helped to disseminate the rudiments of infant hygiene amongst the less enlightened Yiddish-speaking mothers in London and other parts of the world. In 1906 a journal of pædiatrics (*Jikwa Zasshi*) was started in Tokyo, Japan. In the same year was held the first extensive exhibition for infant welfare in Berlin. In 1907, as the result of the investigations of Leslie Mackenzie and of Hay into the physical condition of school children in Edinburgh and Aberdeen respectively, a School Medical Service was established under the Education Act. In 1908 the Children's Act was passed. This Act, for which Sir Herbert Samuel is responsible, may be called the Magna Carta for children, and deals with infant life protection (including registration and supervision of homes where children under 7 are farmed out); prevention of cruelty to children under 16; Reformatory and Industrial Schools, Juvenile Courts, &c. In the same year the L.C.C. (General Powers) Act empowered local authorities in the Metropolis to appoint health

[1] Villiers-le-Duc is a village situated in Côte-d'Or, near where General Foch made his headquarters during the first battle of the Marne. Its population was only 230 in 1891, and was depleted by the war to 186. Between 1894 and 1921 there were three infants' deaths out of 124 births, so that the results of M. Morel's work, though very good, are not so wonderful as they appear at first sight. The regulations introduced by M. Morel de Villiers provided medical attendance before, during and after labour to every expectant mother, whether married or not; sterilized milk for the baby (if not breast-fed), as well as a monetary grant to the mother whose baby was alive and well at the age of one year; an idea which, as we have seen, was suggested by Michael Underwood in 1774 (see p. 47).

visitors and infant consultation centres for the purpose of instructing mothers in the management of children under school age. In 1909 the *Kaiserin Augusta Victoria Haus* for the prevention of infant mortality was opened in Charlottenburg. In 1910 a similar institution was founded in Russia, by K. Rauchfuss, with the object of training physicians, midwives, nurses, and health visitors, as well as of carrying out research in regard to all questions concerning maternity and child welfare. In 1916 Ehrlich suggested the treatment of congenital syphilis in infants by the injection of salvarsan into the veins of the nursing mother, a practice which has been given up because it is impossible to regulate the dose of the drug received by the infant. The medico-sociological journal, *The Child*, was established in London in 1910, under the editorship of Dr. T. N. Kelynack, who, by his own numerous literary contributions, as well as his indefatigable editorial labours, has done most valuable work in connection with the various ramifications of child-welfare activities. In 1910 Finkelstein absolved both the casein and the fat in cow's milk of all blame in the causation of intestinal disorders in infants and laid it on the sugar. The National Health Insurance Acts which, amongst other things, provide maternity benefit, were passed in 1911 and 1913. In 1913 the first open-air nursery school for children between 2 and 5 years old was opened in London as the result of the zeal and propaganda of the sisters Rachel and Margaret McMillan, and in 1918 such schools were placed on the Statute book.

Ophthalmia neonatorum regulations were issued in 1914. These make the disease notifiable.

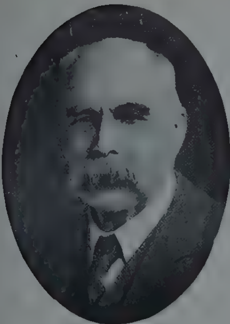
In 1914 the Local Government Board issued a circular offering a grant to local authorities in respect of the following activities.

Ante-natal.—(1) Local supervision of midwives ; (2) antenatal clinics, (3) home visiting of expectant mothers ; and (4) provision of maternity hospitals or beds at a hospital for the treatment of complications of pregnancy.

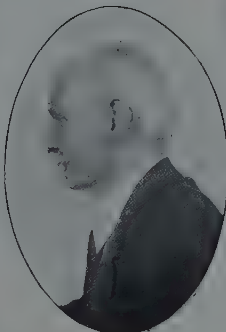
Intra-natal.—(1) Provision of necessary skilful aid to women in labour in their own homes ; (2) provision of beds at a hospital for the treatment of cases of difficult labour.

Post-natal.—(1) Provision of hospital treatment for complications occurring after parturition, whether in the mother or in the infant ; (2) infant clinics ; (3) children's clinics ; (4) systematic home visiting of infants and children.

HELIOTHERAPY AND GENERAL INFANT HYGIENE.



TH. A. PALM.



LEONARD HILL.

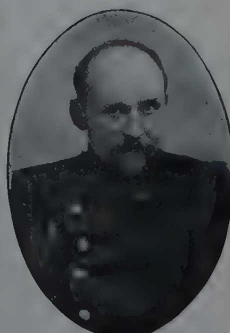
A. ROLLIER.

A. LEVINSON.

I. A. ABT.

L. FINDLAY.

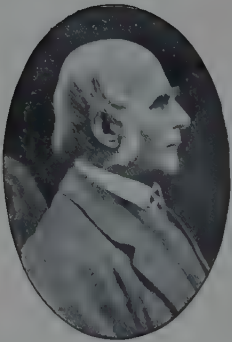
TRUBY KING.



N. P. GUNDOBIN.

PLATE XII.

HEREDITY.



F. GALTON.

G. MENDEL.

F. W. MOTT.

This memorandum may be considered as the real starting point of infant welfare work, of a preventive character, in England.

In 1918 the Maternity and Child Welfare (M. and C. W.) Act was passed, requiring that provision of schemes for maternity and child welfare (for children from birth to 5 years of age) shall apply to all areas. It allows grants for lying-in homes, food for expectant and nursing mothers, homes for children of unmarried, widowed, and deserted mothers. In 1919 the Ministry of Health was established. This Act transferred the administration of the various Statutes dealing with matters of public health from various departments to one minister, e.g., the Midwives Act was transferred to the Ministry of Health from the Privy Council; the Children Act from the Home Office; Schools from the Board of Education, &c. In 1922 the Milk and Dairies (Amendment) Act was passed; this provides for the prohibition of dilution or other adulteration of milk. It prohibits the sale of milk coming from cows with tuberculous udders. The Milk and Dairy (M. and D.) Order, 1923, lays down the standards required for graded milks, such as "certified," "grade A tuberculin tested," "grade A," "grade A pasteurized" and "pasteurized."

In 1925 the Widows,' Orphans' and Old Age Contributory Pensions Act was placed on the Statute book.

In September, 1925, the Fifth Assembly of the League of Nations organised by the Save the Children Fund International Union (founded in 1920) endorsed the *Declaration of Geneva* which, in five clauses, enunciates the rights of children:—

(1) To be fed and cared for in health and disease, whether it was born in or out of wedlock, and whether its parents are alive or dead.

(2) To have an opportunity to develop normally, both physically and mentally.

(3) To be the first to receive help in times of distress.

(4) To be taught a trade, or occupation, which will enable them to earn a living, and to be protected from exploitation.

(5) To be brought up as useful citizens and members of society.

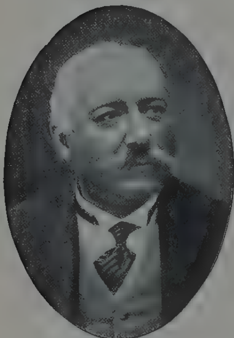
This declaration has been subscribed to by representatives of many different nationalities.

In 1926 the Midwives and Maternity Homes Act was passed, whose main provisions are: (1) The suppression of the practice

of midwifery by the "handy woman." (2) The giving of power to local authorities to form insurance clubs against medical expenses arising from confinement, thus sanctioning the scheme carried out by Dr. William G. Savage, M.O.H. for Somerset, since 1918. (3) Registration of maternity homes.

Scientific research and literary output in the various subjects of child hygiene have been going on at a very rapid rate throughout the twenty-five years of this century. Some of these have already been noted. Bohr and Hasselbach carried out their classical researches on metabolism in the embryo and fœtus in 1903. Their researches were followed up by numerous other investigators, and in 1911 Carpenter and Murlin published their investigations on the metabolism of the human fœtus. In 1908 appeared Gundobin's work on "Peculiarities of Childhood" (in Russian), and in 1912 this work was translated into German (Besondesheiten des Kindesalters) by Dr. S. Rubinstein. In 1913 Bela Schick introduced the now famous Schick test for immunity against diphtheria, and in the same year Behring applied clinically the use of toxin-antitoxin, as suggested by Theobald Smith in 1907, for immunizing against diphtheria all those cases that are positive to the Schick test. The Dick test for immunity against scarlet fever was introduced in 1924. In 1917 there appeared from the pen of the present author an anthropological and sociological study entitled "The Jewish Child, its History, Folklore, Biology and Sociology," and in 1920 the same author published "The Principles of Ante-natal and Post-natal Child Physiology"—so far the only book of its kind. The author also introduced lectures on Child Physiology to the nurses at the Infants Hospital, London, in 1915, post-graduate lectures on Ante-Natal and Post-Natal Child Physiology and Hygiene, at the London Hospital Medical College, in 1921, and public lectures on the same subject under the Chadwick Trust, in 1924. It would be impossible to enumerate all those who have done valuable work in connection with the different phases and aspects of child life throughout the world, but the following, in addition to those already mentioned, are a few outstanding names. Alois Epstein, who was the founder and first director of the world-renowned Foundling Asylum, at Prague, managed to reduce the mortality in that institution from 30 to 5 per cent., by the adoption of rigid aseptic methods. Marfan (editor of *Le Nourisson*) and Nobécourt did original work on infant nutrition. Arthur Schlossmann, Meinhard von Pfaundler, Ludwig Tobler, Thomas Morgan Rotch, Emmet Holt, John Howland,

NEO-NATAL LIFE.



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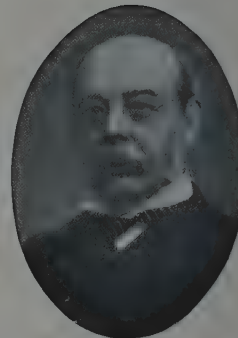
S. G. Moore.



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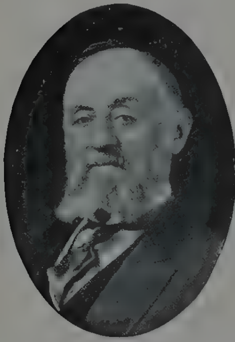
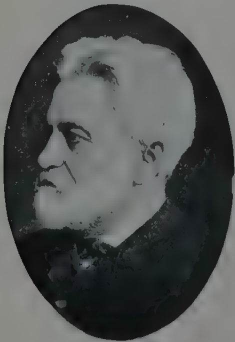


N. Straus.

A. Louise McIlroy.

H. Rothschild.



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A. Ylppö.

P. Budin.

Henry Dwight Chapin, Bert Raymond Hoobler, Alfred and Julius Hess, Luigi Concetti, Yussuf Ibrahim, Arvo Ylppö, George Frederick Still, John Poynton, Edward Mellanby, Harriette Chick, Leonard Findlay, Leonard Hill, Noël Paton, Abraham Levinson, C. W. Saleeby, are names familiar to all who are interested in matters connected with child life. Lastly, the wonderful work of Maria Montessori in the mental development of the pre-school child deserves honourable mention.

Among the large number of workers on the subject of ante-natal metabolism in animals and man during the present century, the following may be mentioned: Jägernoos, Bar and Daurez, Hoffström, Rubner and Langstein; Slemons, Zaccharjewsky, in addition to those previously referred to.

I have in the preceding pages endeavoured to epitomize the history of the movement for infant and child welfare from the times of the earliest recorded antiquity to the present day. The sketch is necessarily a very brief and imperfect one, but it will, I hope, suffice to convince the reader of the truth of Bartholini's remark: "Pessime enim studiis suis consulunt, qui ita retentiorum scriptis se immergunt ut veteres vel negligant, vel contemnant quum plerarumque rerum lux ex illis pendeat."

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CHAPTER III

THE BIOMETRICS OF CHILD HYGIENE

"He who will not reason is a bigot; he who cannot, is a fool; and he who dare not, is a slave."—BYRON.

BEFORE dealing with the actual numerical data in connection with the vital statistics of early life and other matters to be considered in subsequent chapters, it is necessary to discuss briefly a few of the constants employed by modern scientific statisticians, and to point out some of the pitfalls which must be avoided by the student of child hygiene in his attempt to draw conclusions or otherwise make practical use of the statistical data that he may extract from blue books or other sources. "The day has passed," says Raymond Pearl, "when the mere tabulation of crude statistics and the drawing of unchecked and uncriticised conclusions from the tabulations will pass the court of scientific judgment. No statistical conclusion is to-day accepted by competent workers in the field unless it is fortified by a determination of the probable error involved. . . . One may hope that as time goes on a higher and more critical standard than now prevails will become the rule instead of the exception in statistical operations and deductions in public health work."

It is often alleged by the uninitiated that "it is possible to prove anything by statistics." Indeed, it has been said that there are three degrees of inexactitude: lies, dreadful lies, and statistics. This of course is altogether untrue. Statistics are merely facts expressed by numbers, and facts do not become fiction when expressed numerically. If statistics are properly collected and logically used they are bound to lead to the right conclusion, and when different conclusions are drawn from the same statistics the fault does not lie with the statistics but with the manner in which they have been collected or employed. Every schoolboy knows that it is possible to "prove" by elementary mathematics that "one and one are three," yet nobody would condemn mathematics as unreliable, because by some clever ruse mathematics can be made to lead to an obvious absurdity.

Statistical Methods.—It would be impossible to enter into a detailed description of the modern methods of statistical analysis.

The reader will find all the information he requires in the author's book on "Biomathematics," where a special chapter is devoted to **Biometrics** or the application of statistical methods to biological science. It will suffice in this place to explain, by means of a few illustrations, the meaning of certain terms commonly used by statisticians and to point out their importance in relation to problems of child hygiene. An attempt will also be made to explain certain fallacies that must be avoided in the use of statistics.

Arithmetical Mean or Average.—Supposing we have a number of measurements, such as the heights of several boys of the same age, or the rates of mortality of infants during a consecutive series of years, and so on. We generalise the results of each of the sets of such observations by giving a single figure as the type of the entire group. Such a representative figure is called an average, of which there are several kinds. The one most commonly used is the **Arithmetical Average** or **Mean**, which is found by dividing the sum of the magnitudes of the various measurements by the number of such measurements. Thus, the infant mortality rates for the years 1916-20 were 91, 96, 97, 89, 80, respectively, and hence the average or mean infantile mortality during the quinquennium in question was

$$\frac{91 + 96 + 97 + 89 + 80}{5} = \frac{453}{5} = 90{\cdot}6$$

Standard Deviation.—Whilst the average gives us a representative figure typical of an entire group or array of numbers, it gives us no idea of the *variability* of the numbers out of which the average has been obtained. We cannot, by merely looking at the average, say whether the component numbers of which it is the resultant differ from one another much or little. Thus, supposing we have the following two sets of measurements representing mortalities at a certain age for a consecutive number of years, in two different parts of the world, A and B.

Year	A	B
1911	28	15
1912	22	25
1913	27	36
1914	23	19
1915	30	35
Average	26	26

The average or mean mortality for A $= \frac{28+22+27+23+30}{5} = \frac{130}{5} = 26$, and the average mortality for B $= \frac{15+25+36+19+35}{5} = \frac{130}{5} = 26$, so that the mean is the same for both places, but the *variability* or the *dispersion* of the different figures about the mean is different in the two cases. Thus, the greatest deviation from the mean in the case of A is $30 - 26 = 4$, whilst in the case of B it is $15 - 26 = -11$. It is therefore necessary to know also the average or mean deviation of the individual measurements from the general mean. Thus, in the above cases the various deviations (above or below the mean) are, in the case of A, $+2$ ($= 28 - 26$), $-4, +1, -3, +4$, and in the case of B, $-11, -1, +10, -7, +9$, so that the average deviation from the mean, neglecting signs, is $\frac{14}{5} = 2{\cdot}8$ in the one case, and $\frac{38}{5} = 7{\cdot}6$ in the other. We know, therefore, by stating the average deviation from the mean, which of the two sets of measurements is the one which shows greater variability. For many reasons, however (see "Biomathematics," p. 334), the average deviation is not much utilised in statistical work, and one uses instead a number which is called the **standard deviation.** This is obtained by squaring the individual deviations, dividing the sum of these squares by the number of deviations, and taking the square root of the quotient. Thus, in the cases under consideration we get the standard deviation for

$$A = \sqrt{\frac{2^2 + 4^2 + 1^2 + 3^2 + 4^2}{5}} = \sqrt{\frac{46}{5}} = \pm 3{\cdot}03,$$

whilst in the case of B, the standard deviation

$$= \sqrt{\frac{11^2 + 1^2 + 10^2 + 7^2 + 9^2}{5}} = \sqrt{\frac{352}{5}} = \pm 8{\cdot}39.$$

As the standard deviation measures the degree of concentration of the various deviations around the mean, it is clear that it also measures the precision of the observer; the greater the standard deviation in a series of observations the less the precision with which the observations were made.

The standard deviation is generally abbreviated into S. D., or is represented by the Greek letter σ, and is a constant of great value in modern statistical work. Its generic

formula is $\sigma = \sqrt{\frac{\Sigma d^2}{n}}$, where Σd^2 stands for the sum of the squares of the individual deviations and n = number of deviations.

Coefficient of Variation.—The percentage ratio between the standard deviation σ and the arithmetical mean, A.M., is called the coefficient of variation, and is a constant which is used for the purpose of comparing the variability of different sets of measurement. To take a concrete example: The neo-natal mortality (i.e., the mortality of infants during the first month of life) in forty-seven different cities in the same country ranged between 34 and 54 per 1,000 live births during the same year, with an average of 44 and a standard deviation of 5·2. In the same cities, in the same year, the mortality of infants between the second and twelfth months of life ranged between 22 and 86, with an average of 50 and a standard deviation of 14·8. Which is the more variable rate?

Here it is wrong to say that merely because the standard deviation is greater in the second case therefore the variability of later infantile mortality is greater, because the average in the two cases is not the same. But, by taking the coefficient of variability, we get

$$\text{C. of V. (1)} = \frac{100\,\sigma_1}{AM_1} = \frac{520}{44} = 12 \text{ per cent. of its mean.}$$

$$\text{C. of V. (2)} = \frac{100\,\sigma_2}{AM_2} = \frac{1480}{50} = 30 \text{ per cent. of its mean.}$$

Hence the later infantile mortality is two and a half times as variable as neo-natal mortality.

The Probable Error (P.E.).—No measurement, however carefully made, is ever absolutely exact, and therefore the constants derived from these measurements must necessarily also fall short of absolute exactness.

Again, if we were to take different random samples of the same thing with whose measurements we are concerned, we would find that the means of each of these samples, although perhaps approximately the same, are not as a rule exactly so. For example, if we take 100 boys of the same age and divide them indiscriminately into five groups of twenty, we would find that the mean heights of each of the five groups are approximately but not exactly the same, and that each of these means would probably be different from the mean height of all the 100 boys measured together. This fluctuation in the results may be

due to a number of factors, e.g., racial composition of the group, its social position, &c., but one of these is the element of chance or of random sampling. Hence it is necessary in every case to ascertain, if possible, the precise degree of reliability that can be attached to any particular constant or conclusion derived from statistical data, i.e., we must ascertain by how much the constant in the random sample differs from the *true* value of the constant if the sample were to contain all the possible individuals. This is done by finding what is called the *probable error*, and the figure representing this probable error is put after that which represents the mean or any other statistical constant under consideration. Thus, during a given year the infant mortality in a certain area was found to be 104·49, with a probable error of 1·78. This is recorded as follows: Infant mortality = 104·49 ± 1·78, and it means that the probability of the true infant mortality rate (i.e., the mean infant mortality as determined from an infinite number of cases) lying between 102·71 (i.e., 104·49 − 1·78) and 106·27 (i.e., 104·49 + 1·78) is exactly the same as its being either below 102·71 or above 106·27.

Factors influencing the Probable Error.—It is obvious that one of the factors that enters into the reliability of a given statistical constant must be the number of observations upon which the constant is based. Thus, a mortality rate based upon, say, 100 cases, is not so reliable as one based upon 1,000 cases, and the latter is less reliable than one calculated from 100,000 cases. It can be shown mathematically that the probable error varies inversely as the square root of the size of the statistical sample. Thus, if the size of the sample is 100, the probable error of the mean, for instance, would be ten times as great as that derived from a sample of 10,000, because $\sqrt{10{,}000} : \sqrt{100} = 10 : 1$.

The probable error of the standard deviation of a normal distribution $= \frac{0·6745\,\sigma}{\sqrt{2n}}$ and that of the coefficient of variation is $\frac{0·6745\,\sigma}{\sqrt{2n}} \left\{1+2\left(\frac{\sigma}{100}\right)^2\right\}^{\frac{1}{2}}$.

Another factor upon which the probable error depends is the standard deviation: the greater the S.D. the greater the probable error, since, as we have seen on p. 66, the S.D. is a measure of precision of observations. It can be shown that the probable error of the mean is given by the formula $\frac{0·6745\,\sigma}{\sqrt{n}}$, or about $\frac{2\,\sigma}{3\sqrt{n}}$ where σ = standard deviation and n = number of observations (see p. 92).

Theoretical considerations (see p. 92) show that if a measurement is less than three times its probable error (or, which is the same thing, less than twice the standard deviation, since P.E. $= \frac{2}{3}\sigma$), then that measurement is of no great statistical significance. If, however, it is equal to or greater than three times the probable error, then its significance is very considerable, and increases in proportion to the number of times the measurement is greater than P.E. Thus, if the measurement $= 3$ P.E. or $2\,\sigma$, then the probability of the measurement being the true value is greater than 49 : 1 (see p. 92). If the measurement is four times the probable error then the probability of its being correct is about 99 : 1.

Probable Error of a Difference.—Supposing we have two measurements of a certain constant, such as a mean, derived from two separate samples, and we wish to ascertain whether the difference in the size of these constants is due simply to errors inherent in random sampling or to real differences in the characters of these samples. In the case of mortality, for instance, the age distribution, racial composition, social status, &c., might enter into consideration; we again determine the probable error of that difference. The theory of probability tells us that the probable error of the sum or difference of two quantities, *which are quite independent of each other*, is equal to the square root of the sum of the squares of the quantities under consideration. If the difference in the values of the constant is found to be greater than three times the probable error thus determined, then it becomes significant and cannot be considered as due to errors associated with mere chance or random sampling.

To illustrate the working of the method and its importance, we shall take the following example. The mean infantile mortality rates in a given area during the years 1916, 1917 and 1918 were as follows:—

$$104{\cdot}23 \pm 1{\cdot}75;\ 99{\cdot}24 \pm 1{\cdot}32;\ 111{\cdot}61 \pm 1{\cdot}66.$$

Are we justified in saying that these fluctuations were due merely to random sampling or to some environmental factors?

If we were given the means alone without their probable errors we would not be in a position to answer the question, but being given the probable errors we proceed as follows:—

$$\text{Difference in rates between 1916 and 1917} = 104{\cdot}23 - 99{\cdot}24 = 4{\cdot}99$$

$$\text{Probable error of difference} = \sqrt{(1{\cdot}75)^2 + (1{\cdot}32)^2} = \pm\, 2{\cdot}19$$

Therefore the difference (4·99) between the means is less than three times its probable error ($3 \times 2·19 = 6·57$) and is therefore not significant. On the other hand, in the case of the years 1917 and 1918, we get

$$111·61 - 99·24 \pm \sqrt{(1·32)^2 + (1·66)^2} = 12·37 \pm 2·12,$$

so that the difference is greater than three times its probable error and is therefore significant. In other words, while the fall in mortality rate between 1916 and 1917 can be explained as due to the operation of chance alone, its rise between 1917 and 1918 must be due to some environmental factor.

Let us take another example. The mean weight of infants at birth, as calculated from the Report of the Anthropometric Committee of the British Association, 1883, is:—

Males: 3·230 ± 0·016 kg.
Females: 3·151 ± 0·015 kg.

For the purpose of studying gaseous metabolism in infants, 51 male and 43 female infants were taken whose mean weights were:—

Males: 3·459 ± 0·0430 kg.
Females: 3·336 ± 0·0564 kg.

Do the nutrition laboratory babies represent a fair random sample of normal infants as judged by the B.A. standard?

Proceeding as above, we have:—

For males: Difference

$$= 3·459 - 3·230 \pm \sqrt{(0·043)^2 + (0·016)^2} = 0·229 \pm 0·046$$

For females: Difference

$$= 3·336 - 3·151 \pm \sqrt{(0·0564)^2 + (0·015)^2} = 0·185 \pm 0·058$$

The difference is in each case more than three times its probable error, and therefore the nutrition laboratory infants are significantly heavier than the normal B.A. standard.

Bernoulli's Method of calculating σ and P.E.

If we have a group of n cases, amongst which the probability of a certain event happening is p, and that of the event not happening is q, then Bernoulli's theorem states that

$$\sigma = \sqrt{npq} \text{ and } \mathbf{P.E.} = \frac{2}{3}\sqrt{npq}.$$

Let us apply this to the following problem. The mean infantile mortality for England and Wales in 1923 was 69 per 1,000 live births. In 1924 it had risen to 74 per 1,000. The total number of births in 1924 was 750,000. Can the rise in mortality during that year be accounted for by operation of

chance or is it due to some external factor? Here $n = 750{,}000$ $p = \frac{69}{1{,}000}$ (i.e., the probability of infants dying); $q = \frac{931}{1{,}000}$ (i.e., the probability of infants surviving).[1]

$$\therefore \sigma = \sqrt{npq} = \sqrt{750{,}000 \times \frac{69}{1{,}000} \times \frac{931}{1{,}000}} = \pm 219{\cdot}5$$

$$\therefore \text{P.E.} = \pm \frac{2}{3}\sigma = \pm \frac{2}{3} \times 219{\cdot}5 = \pm 146{\cdot}3$$

In other words, out of 750,000 births we would expect a fluctuation of 146·3 above or below the expected number of deaths, i.e., a fluctuation of $\frac{146{\cdot}3}{750}$ per 1,000 or approximately 0·2 per 1,000. Therefore the rise is much more than can be explained on the operation of chance alone.

Here is another example given by Newsholme.[2] Two groups of patients of equal age and sex distribution, &c., were treated by two methods, B and C, for the same infectious disease during the same outbreak, and another group, A, in the same circumstances were not so treated. The results were as follows:—

	A	B	C
Number of cases	1,200	865	1,236
Number of deaths	42	20	10
Fatality per cent.	3·5	2·3	0·81

Is there any significant difference between these percentages or might they be the result of random sampling?

Since

$$\sigma_A = \sqrt{\frac{1{,}200 \times 3{\cdot}5 \times 96{\cdot}5}{1{,}200 \times 100 \times 1{,}200 \times 100}} = \sqrt{\frac{3{\cdot}5 \times 96{\cdot}5}{1{,}200}} \text{ per cent.}$$

$$\sigma_B = \sqrt{\frac{865 \times 2{\cdot}3 \times 97{\cdot}7}{865 \times 100 \times 865 \times 100}} = \sqrt{\frac{2{\cdot}3 \times 97{\cdot}7}{865}} \text{ per cent.}$$

$$\sigma_C = \sqrt{\frac{1{,}236 \times 0{\cdot}81 \times 99{\cdot}19}{1{,}236 \times 100 \times 1{,}236 \times 100}} = \sqrt{\frac{0{\cdot}81 \times 99{\cdot}19}{1{,}236}} \text{ per cent.}$$

$\therefore$ In case of groups A and B,
difference in fatality $= 3{\cdot}5 - 2{\cdot}3 = 1{\cdot}2$ % and

$$\text{P.E. of difference} = 0{\cdot}6745 \sqrt{\frac{3{\cdot}5 \times 96{\cdot}5}{1{,}200} + \frac{2{\cdot}3 \times 97{\cdot}7}{865}} = 0{\cdot}5\ \%$$

As 1·2 is less than three times 0·5, it is probably not significant. For A and C, however, the difference in fatality = 3·5 − 0·81 = 2·7 per cent. and P.E. of difference works out as 0·4 per cent., so that the difference is probably significant.

[1] See "Biomathematics," p. 328.

[2] "The Elements of Vital Statistics," London, 1923, p. 512.

Correlation.

One of the most important uses of statistical analysis in biological phenomena is for the purpose of studying the inter-relationship or **correlation** between sets of phenomena. General impressions may tell us that a certain phenomenon in some way depends upon another, but mathematical statistics furnishes us with a method of measuring the degree of interdependence between the two phenomena in question and enables us to compare the degrees of inter-relationship between different variables. For example, we know that infantile mortality in some way depends upon overcrowding. The *coefficient of correlation* between overcrowding and infant mortality gives a quantitative expression of the degree of this inter-relationship.

If two phenomena are entirely independent of each other, we say that the coefficient of correlation between them is zero. If the inter-relationship is perfect, we say that the coefficient of correlation is $\pm$ 1, according as the increase of one perfectly corresponds with the increase or the decrease of the other. Hence the range of a correlation coefficient is between 0 and ± 1. In biological phenomena there is practically no such thing as perfect correlation, so that the coefficient is always some decimal fraction, positive or negative.

It would be beyond the scope of this work to enter into a full description of the method of determining the correlation coefficient between two variables, but a couple of illustrative examples should make the method clear.

The following table gives the weights of and amount of heat produced per hour of a number of new-born infants. In columns 4 and 5 we give the deviations of the weights and heat productions from their respective means. In columns 6 and 7 we give the squares of these respective deviations. In column 8 we give the products of the separate deviations.

The fact that the respective deviations in columns 4 and 5 have always the same sign, so that a baby above the average weight produces more than the average amount of heat and vice versa, in itself shows that there is a correlation between these two variables w and h.

But in order to ascertain the closeness of interdependence between these two variables, we multiply the respective deviations together, as in column 8. The mean of these products, 0·1928, gives us the "mean product deviation." If now we divide this mean product by the product of the two standard

deviations, σ_w, σ_h, the result is the coefficient of correlation between weight and heat production.

1	2	3	4	5	6	7	8
Number	Weight in kgm. (w)	Heat production in calories per hr. (o)	Deviation of wt. from mean dw	Deviation of heat production from mean dh	Square of dw	Square of dh	Product of dw and dh, i.e., dw×dh
8	3·48	6·66	+0·15	+0·45	0·0225	0·2025	+0·0675
10	3·45	6·75	+0·12	+0·54	0·0144	0·2916	+0·0648
30	3·33	6·00	±0·00	−0·20	0·0000	0·0441	0·0000
51	3·73	6·41	+0·40	+0·20	0·1600	0·0400	+0·0800
53	2·87	5·95	−0·46	−0 26	0 2116	0·0676	+0·1196
76	3·56	6·37	+0·23	+0·16	0·0529	0·0256	+0·0368
74	3·63	6 50	+0 30	+0·29	0·0900	0·0841	+0 0870
4	3·28	5·79	−0·05	−0·42	0·0025	0·1764	+0·0210
9	4·04	7·42	+0·71	+1·21	0·5041	1·4641	+0·8591
13	3·25	5·75	−0·08	−0·46	0 0064	0·2116	+0·0368
21	2·92	5·67	−0·41	−0·54	0·1681	0·2916	+0·2214
34	2·90	5·58	−0·43	−0 63	0·1849	0 3969	+0·2709
43	3·62	6·87	+0·29	+0·66	0·0841	0·4356	+0·1914
65	2·63	5·29	−0·70	−0·92	0·4900	0.8464	+0·6440
Mean	3·33	6·21	0·309	0·492	0·1422	0·3270	=0·1928

$$\text{Now } \sigma_w = \pm\sqrt{\cdot 1422},\ \sigma_h = \pm\sqrt{0\cdot 3270}$$

$$\therefore\ \sigma_w\ \sigma_h = \sqrt{\cdot 1422 \times \cdot 327} = \cdot 2151.$$

∴ Coefficient of correlation between weight and heat production, written as $r_{wh} = \frac{\cdot 1928}{\cdot 2151} = 0\cdot 896$.

To this must be added the probable error whose formula is—

$$\frac{0\cdot 6745\ (1 - r^2)}{\sqrt{n}}$$

$$\therefore\ r_{wh} = 0\cdot 894 \pm \frac{0\cdot 6745\ (1 - 0\cdot 894^2)}{\sqrt{14}}$$

$$= 0\cdot 894 \pm 0\cdot 0362.$$

(When a larger number of cases is taken it is found that $r_{wh} = 0\cdot 7833 \pm 0\cdot 023$.)

Similarly it can be shown that the coefficient of correlation between surface areas and heat production varies with the formula used for calculating surface areas, i.e., whether Meeh's formula ($S = 12\cdot 3\ \sqrt[2]{W^3}$), Lissauer's formula ($S = 10\cdot 3\ \sqrt[3]{W^3}$), or the Dubois formula ($S = 71\cdot 84\ W^{0\cdot 425}, H^{0\cdot 725}$) is used.

Thus for Meeh's formula $r_{hsM} = 0\cdot 892 \pm 0\cdot 0358$.

,, Lissauer s ,, $r_{hsL} = 0\cdot 894 \pm 0\cdot 0362$.

,, Dubois ,, $r_{hsD} = 0\cdot 756 \pm 0\cdot 6772$.

The method just outlined of finding the correlation coefficient is that known as the "*product-moment*" method.

Examples: (1) Harris and Benedict have found—

r_{wh} for male infants to be $0{\cdot}7520 \pm 0{\cdot}0411$,
r_{wh} ,, female ,, ,, $0{\cdot}8081 \pm 0{\cdot}0357$.

Is the difference significant?

$$\text{Difference} = {\cdot}0561 \pm \sqrt{0{\cdot}6411^2 + 0{\cdot}0357^2}$$
$$= {\cdot}0561 \pm 0{\cdot}0544,$$

so that the probable error is almost as large as the difference and is therefore of no significance. We can say therefore that the correlation is about the same for boys and for girls.

(2) The infant mortality rates as well as the degrees of overcrowding in twenty-nine London districts have been published in vol. xxiii of "London Statistics." From these the deviations from the mean values of each of these phenomena have been calculated and the standard deviations for infant mortality and overcrowding (σ_m and σ_o respectively), as well as the mean product deviation (p. 72), have been computed.

They have been found to be as follows:—

$$\sigma_m = 20{\cdot}9 \qquad \sigma_o = 9{\cdot}3$$
$$p = 133{\cdot}8$$

What is the correlation coefficient between overcrowding and infant mortality?

We have—

$$\sigma_{om} = \frac{p}{\sigma_o \times \sigma_m} = \frac{133{\cdot}8}{9{\cdot}3 \times 20{\cdot}9} = 0{\cdot}69$$

$$\text{The probable error} = \frac{0{\cdot}6745\,(1 - 0{\cdot}69^2)}{\sqrt{29}}$$
$$= \frac{0{\cdot}6745 \times 0{\cdot}524}{5{\cdot}385} = \frac{0{\cdot}3534}{5{\cdot}385} = {\cdot}065$$

$\therefore$ Full correlation coefficient $= 0{\cdot}69 \pm 0{\cdot}065$.

Put in the algebraical form, the correlation coefficient between x and y is given by—

$$r_{xy} = \frac{\Sigma\,(d_x, d_y)}{n\sigma_x, \sigma_y}$$

Where d_x and d_y are the deviations of any observations of x and y from their respective means and $\Sigma\,(d_x, d_y)$ represents the "mean product deviation."

Another notation used is as follows :—

If x is any variable
and y is any other variable
then $\bar{x}$ represents the mean of the different values of x
and $\bar{y}$ represents the mean of the different values of y
∴ $(x-\bar{x})$ represents the deviation of any value of x from the mean (i.e., $= d_x$)
and $(y-\bar{y})$ represents the deviation of any value of y from its mean (i.e., $= d_y$);
so that the above formula becomes

$$r_{xy} = \frac{\Sigma\,[(x-\bar{x})\ (y-\bar{y})]}{n\,\sigma_x\,.\,\sigma_y}$$

Prediction or Regression Equations.

It can be shown that

If x represents a particular value of any variable,
and $\bar{x}$ represents the value of the mean of that variable.

Also if y represents the corresponding value of another variable which is correlated with x,
and $\bar{y}$ represents the mean value of this variable,

then $y - \bar{y} = r_{xy}\,\dfrac{\sigma_y}{\sigma_x}(x - \bar{x})$,

and $x - \bar{x} = r_{xy}\,\dfrac{\sigma_x}{\sigma_y}(y - \bar{y})$,

so that if we know the value of x we can calculate the average value of the associated character for that particular value of x, and vice versa.

Thus, in the case of weight and heat production we have

$$h - \bar{h} = r_{wh}\,\frac{\sigma_h}{\sigma_w}\,(w - \bar{w}).$$

As $h = 6{\cdot}21$, $\bar{w} = 3{\cdot}33$, $r_{wh} = 0{\cdot}894$
$\sigma_h = 0{\cdot}57$, and $\sigma_w = 0{\cdot}38$,

the equations reduce themselves to

$$h - 6{\cdot}21 = 0{\cdot}894 \times \frac{0{\cdot}57}{0{\cdot}38}(w - 3{\cdot}33)$$

$$w - 3{\cdot}33 = 0{\cdot}894 \times \frac{0{\cdot}38}{0{\cdot}57}(h - 6{\cdot}21),$$

or $h = 1{\cdot}341\,w + 1{\cdot}744$ . . . . . . (1),
and $w = 0{\cdot}529\,h + 0{\cdot}045$ . . . . . . (2).

Examples in Regression.

(1) To illustrate the use of such equations, let us ask ourselves what we would expect to be the average hourly heat production of an infant weighing 3 kg.

Putting w = 3, we get h = 1·341 × 3 + 1·744 = 5·77 calories per hour. This means that if a number of infants weighing 3 kg. each were taken at random their *average* hourly heat production would be found to be 5·77 calories.

Also the equation tells us that a difference of 100 grm. in weight between two infants means a probable difference of 0·134 calories in their hourly heat output, or of 3·12 calories per day.

(2) The regression equation giving the average infant mortality y, for districts in which the extent of overcrowding, x, is known, is

$$y - 126{\cdot}03 = r \frac{\sigma_y}{\sigma_x} (x - 17{\cdot}86)$$

$$= \frac{(0{\cdot}69)(20{\cdot}9)}{9{\cdot}3} (x - 17{\cdot}86) \text{ (see p. 74)}$$

$$\text{i.e.}\quad y = 1{\cdot}55\,x + 98{\cdot}4$$

$$\text{Similarly, } x = 0{\cdot}31\,y - 21{\cdot}0$$

Hence in Stepney, where the percentage of overcrowding during the year in question was 35, we would have

$$y = 1{\cdot}55 \times 35 + 98{\cdot}6 = 152{\cdot}85$$

i.e., the *expected* infant mortality in Stepney in that year is 142·85. In point of fact it was 144.

(3) The correlation coefficient for height between fathers and sons is known to be 0·514. The mean height of these fathers was 67·68 in. with S.D. = 2·70 in. and the mean height of their sons was 68·65 in. with S.D. = 2·71 in. What would one expect to be the mean height of sons whose fathers were 70 in. tall?

Let x = height of fathers
y = height of sons

Then the regression equation for sons becomes

$$y = 68{\cdot}65 = 0{\cdot}514 \times \frac{2{\cdot}71}{2{\cdot}70} (x - 67{\cdot}68)$$

$$= 0{\cdot}516\,x - 34{\cdot}92$$

$$\text{But}\quad x = 70$$

$$\therefore\quad y = 68{\cdot}85 = 0{\cdot}516 \times 70 - 34{\cdot}92$$

$$= 36{\cdot}12 - 34{\cdot}92$$

$$= 1{\cdot}20$$

$$\therefore\quad y = 68{\cdot}65 = 1{\cdot}20$$

$$= 69{\cdot}85,$$

i.e., fathers whose height is 2·32 in. above the average have sons whose height is on the average only 1·2 in. above the general average, i.e., there is a *regression* towards the general mean of all

sons. This explains the origin of the term *regression* equation. But it is clear that such equations may also be called prediction equations, because they enable us to predict the average value of any particular character for any given value of its associated character.

The probable error of any predicted mean value of x is given by

$$0{\cdot}6745\,\sigma_x\,\sqrt{1-r^2}$$

Partial Correlation.—The correlation that we have so far studied concerned itself only with the measurement of the degree of interdependence between one variable, x, and another variable, y, without taking into consideration the possible inter-relationship between this second variable y and other variables. Thus we know that heat production is correlated with stature, but when we say that the correlation coefficient between heat production and stature is 0·6149, we do not take into consideration the fact that stature is also correlated with weight, and that therefore the correlationship between heat production and stature may be due entirely to the inter-relationship between stature and weight. Similarly, we know that infant mortality is correlated with poverty—but we also know that poverty is closely associated with artificial feeding, high birth-rate, dirt, overcrowding, &c., and the question quite naturally arises whether the correlation between infant mortality and poverty may not be due entirely to the correlationship between poverty and those other conditions which we have just enumerated. Correlation, when considered from the point of view of interdependence between one variable and another, without reference to the possible association between the second variable and any one or more other variables, is called *multiple* or *gross correlation*. In order to find out whether the correlation between x and y is not due to the correlation between y and other variables, we apply the method of *partial* or *net* correlationship by eliminating the effect on x of those other variables with which y is correlated. Thus, in the case of heat production, in order to ascertain whether or not the correlationship between heat production and stature is due to the dependence of weight upon stature, we must determine the partial or net correlationship between heat production and stature for constant body weight. If the correlationship between heat production and stature is merely the result of the dependence of weight upon body length, then when weight is kept constant the correlationship between heat production and stature

should obviously be zero. Similarly, if by eliminating the influence of artificial feeding and the other concomitants of poverty we still find that the correlationship between infant mortality and poverty remain the same, we say that there is a net or partial correlation between infant mortality and poverty *per se.*

The formula used for partial correlationship is the following :—

$$_z r_{zy} \text{ or } r_{xy.z} = \frac{r_{xy} - r_{yz} \cdot r_{xz}}{\sqrt{1 - r_{yz}^2}\ \sqrt{1 - r_{xz}^2}}$$

in which $_z r_{xy}$ or $r_{xy.z}$ is read "the correlation coefficient between x and y for constant z, or when the effect of z upon y is eliminated."

To take a numerical **example** :—

Let r_{hs}, or correlation coefficient between heat production and stature, be 0·6149.

Let r_{sw}, or correlation coefficient between stature and body weight be 0·5725, and let r_{hw}, or correlation coefficient between heat production and body weight, be 0·7960, then

$$\begin{aligned} _w r_{hs} \text{ or } r_{hs.w} &= \frac{r_{hs} - r_{sw} \cdot r_{hw}}{\sqrt{1 - r_{sw}^2}\ \sqrt{1 - r_{hw}^2}} \\ &= \frac{0{\cdot}6149 - 0{\cdot}5725 \times 0{\cdot}7960}{\sqrt{1 - 0{\cdot}5725^2}\ \sqrt{1 - 0{\cdot}7960^2}} \\ &= \frac{0{\cdot}1592}{0{\cdot}8199 \times 0{\cdot}6052} \\ &= \frac{0{\cdot}1592}{0{\cdot}4962} \doteq 0{\cdot}321 \end{aligned}$$

The probable error of this partial correlation coefficient is

$$\frac{0{\cdot}6745\,(1 - r_{hs.w}^2)}{\sqrt{n}}$$

In the example chosen n happens to have been 136.

$$\therefore \text{ P.E.} = \frac{0{\cdot}6745\,(1 - 0{\cdot}321^2)}{\sqrt{136}} = 0{\cdot}0519$$

$$\therefore r_{hs.w} \text{ or } {_w r_{hs}} = 0{\cdot}321 \pm 0{\cdot}0519$$

As the value of this net coefficient is much more than three times its P.E., we conclude that there is some definite correlation between heat production and stature as such. On the other hand, the fact that the net coefficient is only about half the gross coefficient between heat production and stature, we infer that the correlation between stature and body weight to a large extent

determines that between heat production and stature. It is to be remembered, however, that stature is correlated not only with weight but also with age. Allowing also for age (a) we get

$$r_{hs.wa} = 0{\cdot}2899 \pm 0{\cdot}0530$$

We see, therefore, that as we eliminate the various factors which have a relationship with stature, the net correlation between stature and heat production gradually diminishes and tends to become insignificant in proportion to its probable error.

If, instead of determining the net correlation between heat production and stature, we calculate the net coefficient for heat production and weight after eliminating the effect of stature and age, we find $r_{hw.sa} = 0{\cdot}7510 \pm 0{\cdot}0252$. This does not differ materially from the gross coefficient r_{hw} which $= 0{\cdot}7960 \pm 0{\cdot}0212$. Hence we see again that stature in itself has practically no influence on metabolism.

In the case of new-born infants (where age is of course constant)

$$r_{hs} = 0{\cdot}6848 \pm 0{\cdot}0369$$

$$r_{sw} = 0{\cdot}821 \pm 0{\cdot}023$$

$$r_{hw} = 0{\cdot}7833 \pm 0{\cdot}0269$$

From this we get

$$r_{hs.w} = \frac{0{\cdot}6448 - 0{\cdot}821 \times 0{\cdot}783}{\sqrt{1 - 0{\cdot}821^2}\sqrt{1 - 0{\cdot}783^2}} = 0{\cdot}0155$$

This is an utterly insignificant correlation coefficient.

Hence we see that when the inter-relationship between stature and other variables is allowed for, there is no correlation between stature and heat production.

In the same way, it has been shown by Greenwood and Brown that when the influence of artificial feeding and other conditions associated with poverty is accounted for, the net correlation co-efficient between poverty and infant mortality becomes $0{\cdot}111 \pm 0{\cdot}071$, i.e., utterly insignificant.

"The coefficient of correlation," says Professor Whipple, "forms an admirable weapon for exploding false theories," and he quotes the following example. It has been stated recently that statistics show that influenza outbreaks in one year are followed by measles the next year, but such a statement does not stand the test of correlation. Thus, Whipple finds that the correlation coefficient between the two phenomena is $0{\cdot}43 \pm 0{\cdot}16$, so that the coefficient is less than three times its probable error and is therefore of very little significance.

The beginner might find it difficult to understand why it is that we go in a roundabout way to determine by mathematical formulæ the net correlationship between two variables and do not proceed directly to determine by the ordinary method adopted in the case of gross correlation, but choosing the material in such a way that the only variables present are those between which we seek to ascertain the degree of correlation, such as stature and heat production. The answer is that, as a rule, in the case of any statistical sample under consideration this would be impracticable, even if not altogether impossible, since most samples are as a rule not very large, and if we were to select from such samples individuals that differ only in one particular character, such as heat production alone, the number would become so small as to render any correlation totally unreliable. What the mathematical formulæ for determining partial correlation enables us to do is to calculate from the sample at our disposal the correlation which would be found (within the limits of the probable error of random sampling) if it were possible to select from the material at our disposal a group of individuals that differ only in the two variables, such as height and heat production, without so reducing the number of individuals in the group as to render the correlation untrustworthy.

Correlation and Causation.—From what we have said just now under partial correlation, it will be clear that the mere statement that two phenomena are correlated does not imply that one phenomenon is the cause of the other. Even when we have measured the partial correlation between x and y by eliminating those variables with which y may itself be correlated, we cannot with any certainty say that y is the cause of x, for several reasons. In the first instance it is conceivable that we may have failed to eliminate all the possible variables which are inter-related with y (e.g., see numerical example, p. 78, *et seq.*). Secondly, it is in some cases difficult, if not impossible, to say whether the existence of correlation between x and y shows that y is the cause of x, or x is the cause of y. Thus, it has been stated by those who advocate birth control that because a high infantile mortality is associated with a high birth-rate therefore a high-birth rate is a cause of high infantile mortality. It is just as likely that a high infantile mortality may be the cause of a high birth-rate, since families in which many infants have died will probably have more children to compensate for those they have lost. Thirdly, we may have a high correlation between two variables when for

obvious reasons the two variables cannot possibly be causally connected. Thus, Pearson has found the correlation coefficient for height between husband and wife to be very high, as is also the correlation coefficient for eye-colour and other physical characters between husband and wife, and yet it is obvious that any particular physical character in either marital partner cannot be the direct cause of the same character in the other partner, and such resemblance as exists must necessarily be due to selective mating. Indeed, the business of statistics, as such, is not to establish any causal relationships, but only to establish association between two variables and to measure the degree of that association. The only true method of establishing causation is tho experimental method, by means of which we not only eliminate all the variables which may condition a phenomenon, but we can determine which of the two variables selected is the cause and which is the effect. This, however, is of course generally ruled out in the field of human biology. Thus, we cannot keep, say, two groups of 1,000 pairs of human parents of the same breed in experimental cages under similar environmental conditions, and by regulating the sizes of the families in these two groups determine the effect of the number of births upon the rate of infantile mortality. In the absence of the experimental method, the only substitute for it is the application of the laws of logic to the statistical method. "The task of the statistician," says Westergaard, "is not so much to find the causality himself as to help others to find it."

It must be remembered that although it is true that when two things are equal to a third thing they are equal to each other, it is not true that when two series of events vary as a third event, they are necessarily related to each other. Thus, in the words of Whipple, "Infant mortality increases with atmospheric temperature in summer; the softness of the asphalt pavements increases with the atmospheric temperature in summer; but we cannot infer that there is any relation between infant mortality and the softness of asphalt pavements. The actual connection between events is not shown by statistics or by the statistical methods, except as the data are interpreted according to the laws of logic."

The following illustration, given by Professor Raymond Pearl,[2] shows the fallacy of *post hoc ergo propter hoc* reasoning. In 1881, before the diphtheria bacillus had been discovered, an

[2] "Studies in Human Biology," Baltimore, 1924.

author of standing "proved" that the eating of potatoes was the cause of diphtheria, because (1) diphtheria appeared after the introduction of potatoes as an article of diet; (2) the disease increased with the increased consumption of potatoes; (3) in places where potatoes were consumed less, the incidence of diphtheria was less, and so on. "Now the point I wish to make about this, . . ." says Pearl, "now that we *know* what causes diphtheria, is that the statistical reasoning in the paper cited is every bit as good and as cogent as at least much of the statistical work in the field of public health in this country at the present time. We may be, and I fear too often actually are, making just as egregious spectacles of ourselves in our statistical discussions of tuberculosis, infant mortality, &c., as was the gentleman who proved that potatoes cause diphtheria. I hope that the moral of this true story is plain and will sink deep."

Pitfalls to be Avoided when Dealing with Statistics.

(1) Never set out to look for statistics to *prove* a point. In other words, when dealing with statistics you must not have any preconceived ideas as to what the figures you are investigating are going to prove. Allow yourself to be led by the statistics rather than let the statistics be led by you. "Beware," said a teacher to a student launching out into research work for the first time, "lest you find what you are looking for."

(2) Do not reject any observation simply because its deviation from the average is more than you expect or because it seems contrary to your expectation.

(3) Try to weigh and record *all* the possible causes of an event, and do not attribute to one what is really the result of the combination of several. This has been sufficiently dealt with in the paragraphs describing the use of partial correlation.

(4) Be sure that the data which you are comparing are really comparable. Thus, in comparing infant mortality figures in different places we must be sure that there is no difference in the racial, sex and age distribution of the babies, geographical position of the places, &c., before we can draw any conclusion with regard to the influence of any controllable environmental factor.

The following example will serve as an illustration. In a certain infant welfare clinic the mortality of the supervised babies was 78 per 1,000. The mortality of unsupervised infants outside the clinic was 140 per 1,000. These figures at first sight

seem to show a saving of 62 per 1,000 infants effected by supervision, but such an inference would be a gross exaggeration of the value of supervision, because whereas the supervised babies were all between $5\frac{1}{2}$ and 7 months, those unsupervised were, of course, of all ages between birth and one year, amongst whom the average mortality was 34 per 1,000 higher than amongst those between $5\frac{1}{2}$ and 7 months belonging to the same group. Hence, before making the two groups of supervised and unsupervised babies comparable from a statistical standpoint, account must first be taken of the age distributions of these groups by subtracting 34 from the mortality of the unsupervised. If we do that we get the relative mortalities amongst the supervised and unsupervised as 78 and 106 per 1,000 respectively, so that the saving effected by supervision becomes at once reduced from 62 to 28 per 1,000.

Moreover, 87 per cent. of the supervised babies were breast-fed before enrolment, whilst the percentage of breast-feeding in the whole city was only 80, so that the clinic babies were at an advantage from the beginning—since the mortality of breast-fed infants is about one-third that of those artificially fed. By making this allowance, there results a net saving of 20 per 1,000 instead of the apparent saving of 62 per 1,000.

Many more such examples will be met with in the next chapters.

(5) Beware of drawing conclusions from spatial or temporal differences in the value of a certain variable, until the probable errors of these differences have been measured.

(6) *Errors in the Collection of Statistics.*—Assuming that statistics have been honestly and carefully collected, the errors may be of two kinds, viz., *balanced* errors and *unbalanced* errors. If a weighing machine or height gauge is correct, we can take it for granted that a careful observer will make as many small errors of observation above the correct weight or height as below them, so that in a large number of records of weights and heights the errors will cancel themselves out. Such errors of observation are balanced, or unbiased, errors. On the other hand, if the weighing machine or height gauge is faulty, the errors in the records become unbalanced or biased errors, and special allowance must be made for them. A good example of biased error in vital statistics is the apparently high infantile mortality due to imperfect birth registration (see p. 134).

(7) *Error in Defining the Unit.*—We must be quite sure

about the meaning of such terms as "birth," "infant," "poverty," "overcrowding," &c. Does "birth" refer only to a "live birth," or is a "stillbirth" also to be included? and does it or does it not include a "premature birth," and what do we understand by premature or stillbirth? In some cases it is probable that the definition of the unit has changed with the progress of medicine, and the change has made comparison of statistics either difficult

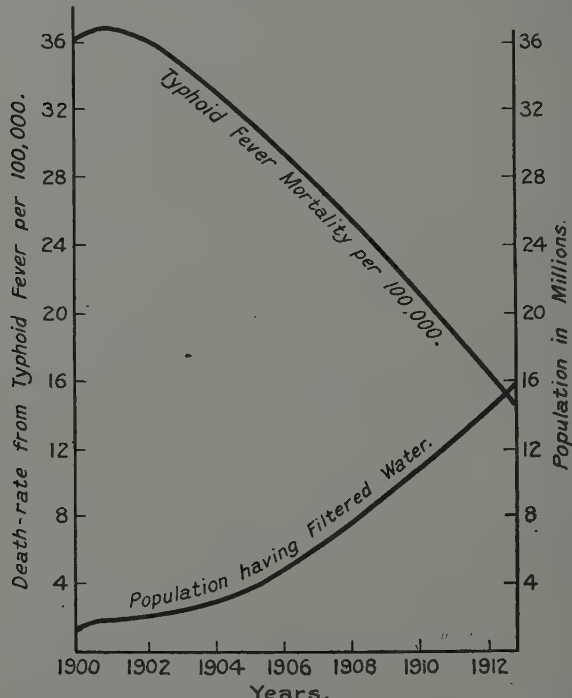

Fig. 21.

or untrustworthy. For example, it is quite certain that with the discovery of the diphtheria, tubercle, and other bacilli, as well as of the Widal and Wassermann tests for typhoid and syphilis respectively, many mild cases that would have been undiagnosed before are now included in the statistics of morbidity and mortality of such diseases, so that it is conceivable that any lowering of mortality or fatality rates from such diseases may be more apparent than real.

(8) *Graphical Pitfalls.*—(*a*) If two variables y and z are plotted as ordinates against a third variable, x, as abscissa, and it is found that there is a close correspondence, either direct or inverse, between the two graphs, so that either they rise and fall together, or, whenever one rises the other falls and vice versa (i.e., one is a mirror image of the other), it must not be at once inferred that there is a direct or inverse causal relationship between y and z. Let the reader bear in mind what we have said in connection with correlation and causation, and he will avoid drawing erroneous conclusions from such graphs. The seasonal distribution of infant mortality, as shown in the chart (fig. 34A), does not imply that either the heat of the summer or the cold of the winter and the rate of infantile deaths are causally related. It is probable that the rise in mortality in the hot summer months is not the direct result of the rise in atmospheric temperature, but is due to some other factor, such as flies, &c., which is in itself dependent upon the atmospheric temperature. Similarly, the winter rise may be due to a number of factors (such as rain, lack of sunshine, &c.) other than the mere fall in temperature. Again, although the reduction in the mortality from typhoid fever is quite certainly due to the purification of public water supplies, this cannot be inferred from the accompanying chart (fig. 21) alone, because the water filtration may quite possibly have been only one of many other factors in the case, viz., mildness of epidemics, better diagnosis of milder cases, better nursing, &c. As a matter of fact, elimination of all other possible causes, as well as direct experimental evidence, conclusively proves the causal relationship between the water filtration and the diminution in the rate of deaths from typhoid fever, but the point that I wish to stress is that the chart alone does not prove it.

(9) A decrease in the incidence of, or mortality from, a certain disease is sometimes plotted with actual rates, *instead of rates of decrease*, as ordinates. In this way one obtains a pictorial representation which gives an utterly erroneous impression of the true state of affairs. Thus, the mortality of infants under 3 months old has decreased from 67 to 43 per 1,000 between the years 1881 and 1923, whilst the mortality of infants between 3 and 6 months during the same period fell from 28 to 10 per 1,000, and the general mortality declined from 19·1 to 11·6 per 1,000. When charted in the form of absolute rates as ordinates, (fig. 22) it appears that the mortality of infants under 3 months old

declined more rapidly than that of infants between 3 and 6 months, and much more rapidly than the general death-rate. As a matter of fact the decline in the first and third cases was about 39 per cent. each, and that in the second 64 per cent. of the original mortality.

In order to obviate such possible errors of interpretation, it is necessary to use ratio charts by taking the original rate in each case as 100 (see figs. 30 and 31), or to use semilogarithmic paper, in which the ordinates are the logarithms of the rates instead of the rates themselves (see fig. 22).

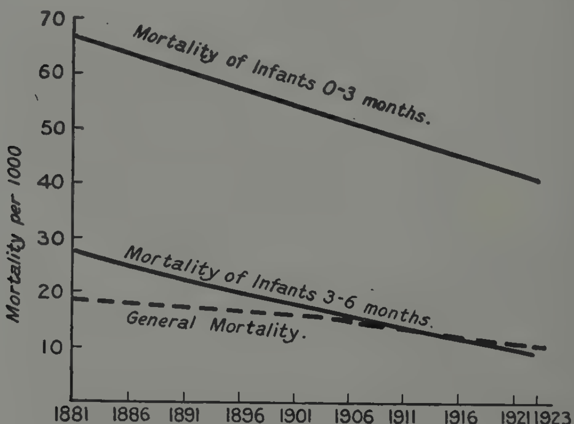

Fig. 22

From what has been described in the foregoing paragraphs, it will be appreciated that the proper interpretation of statistical figures is a matter of vital importance from the point of view of public health. An American writer very aptly describes the relationship between statistical methods and public hygiene as follows: Vital statistics, he says, is the "Cinderella of modern public hygiene sitting in the chimney corner sifting the ashes of dusty figures, while the proud sisters, bacteriology and preventive medicine, go to the ball and talk about the wonderful things they have done."

The Frequency Curve.—No modern student of statistics can afford to be totally ignorant of the use of the frequency curve. Supposing we were to take a random sample of children of the

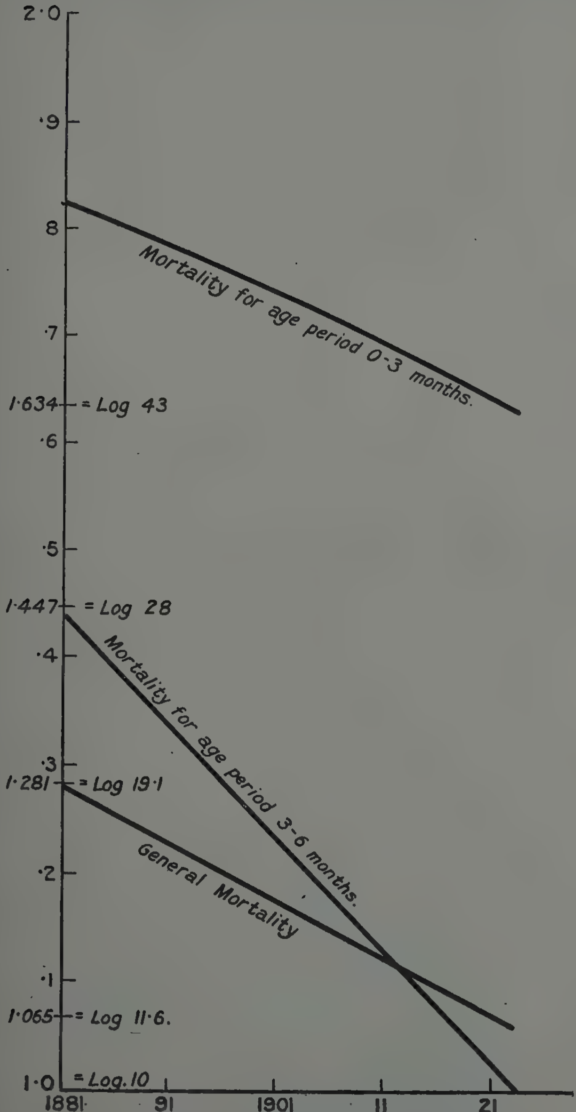
2·0
·9
8
·7
1·634 = Log 43
·6
·5
1·447 = Log 28
·4
·3
1·281 = Log 19·1
·2
·1
1·065 = Log 11·6.
1·0 = Log. 10
1881
91
1901
11
21
Mortality for age period 0-3 months.
Mortality for age period 3-6 months.
General Mortality

FIG. 23.

same age, or of adults, and arrange them in an array in accordance with their heights (i.e., the smallest at one end and the largest at the other), we shall get a curve like that shown in fig. 24. This curve, which is called an ogive, is characterised by the fact that it is nearly flat in the middle but curves down at one end and up at the other. In other words, the individual of average or mean height would be in the middle of the array, *most* of the individuals will approximate in height the mean of them all, a few will differ in height from the mean to a considerable extent, and a very few will differ from it very considerably. Moreover, it will be found, if the sample is large enough, that for each range of deviation from the mean there is

FIG. 24.—Ogive.

an approximately equal number of individuals on either side of the mean; so that if the mean is, say, 67 in., there are as many individuals who are 64, 65 and 66 in. tall as there are 70, 69, and 68 in. respectively. This will be found to hold good not only for heights of individuals, but also (to some extent) for their weights, their intelligence, as measured by the number of marks such individuals would get at an examination, &c. In fact, all the natural phenomena in which the element of *equal* chance predominates are found to be capable of representation by means of such a curve. Thus, in the case of height, the chance of there being an individual, say one inch taller than the mean, is equal to that of there being one shorter by one inch than the mean. The ogive, however, has a limited practical application, because when the number of items is very large, it becomes practically im-

possible to place them in an array. It is then possible to arrange the observations in *groups* and plot the group frequencies as ordinates, with heights as abscissæ (instead of heights as ordinates and the positions in the array as abscissæ, as in the ogive). The resulting curve is called a *frequency curve.* Thus, out of 8,588 adult males in the United Kingdom, it was found that:—

2,	i.e.,	0·02	per cent.,	measured	between	57	and	58	inches
4	,,	0·04	,,	,,	,,	58	,,	59	,,
14	,,	0·15	,,	,,	,,	59	,,	60	,,
41	,,	0·48	,,	,,	,,	60	,,	61	,,
83	,,	0·96	,,	,,	,,	61	,,	62	,,
162	,,	1·92	,,	,,	,,	62	,,	63	,,
394	,,	4·60	,,	,,	,,	63	,,	64	,,
669	,,	7·59	,,	,,	,,	64	,,	65	,,
990	,,	11·53	,,	,,	,,	65	,,	66	,,
1,223	,,	14·25	,,	,,	,,	66	,,	67	,,
1,329	,,	15·48	,,	,,	,,	67	,,	68	,,
1,230	,,	14·25	,,	,,	,,	68	,,	69	,,
1,063	,,	12·38	,,	,,	,,	69	,,	70	,,
646	,,	7·52	,,	,,	,,	70	,,	71	,,
392	,,	4·56	,,	,,	,,	71	,,	72	,,
202	,,	2·35	,,	,,	,,	72	,,	73	,,
79	,,	0·92	,,	,,	,,	73	,,	74	,,
32	,,	0·37	,,	,,	,,	74	,,	75	,,
16	,,	0·19	,,	,,	,,	75	,,	76	,,
5	,,	0·06	,,	,,	,,	76	,,	77	,,
2	,,	0·02	,,	,,	,,	77	,,	78	,,
Total 8,588		100							

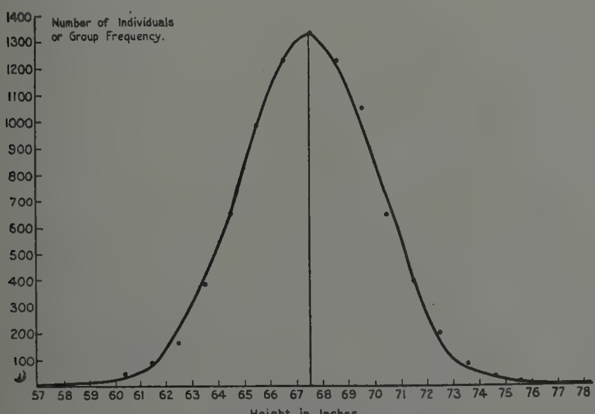

FIG. 25.—Normal frequency curve.

When these actual or percentage frequencies are plotted as ordinates with height intervals as abscissæ, we get the curve shown in fig. 25.

As I have remarked, all those natural phenomena which depend for their occurrence upon the operation of the multiplicity of uncontrollable causes which we call *chance*, are found to be capable of being represented by some sort of a frequency curve. If the chance of the event happening is equal to that of its not happening, then the resulting curve is the *normal* or symmetrical *frequency curve.* Thus, in the case of heights of individuals, some of the numerous uncontrollable causes constituting the even chance that any individual will be shorter or taller by a specified number of inches than the mean of the general population are: heredity, race, social position, occupation, pose of body, physical health, nature of environment with regard to food, air, sleep, sunshine, &c., during the growth period, physical training, &c.

Mathematical Treatment of Frequency Curve.—The normal frequency curve lends itself easily to mathematical analysis and treatment. Indeed, it can be shown that the ordinates of such curve are proportional in height to the various terms of the expansion $\left(\frac{1}{2} + \frac{1}{2}\right)^n$ when n is infinitely large and situated at equal distances along the abscissa.

Properties of Normal Frequency Curve.

(1) It is absolutely symmetrical about the mean ordinate.
(2) It is entirely situated above the x axis.
(2) Its equation is

$$y = \frac{1}{\sigma\sqrt{2\pi}} e^{-\frac{x^2}{2\sigma^2}}$$

Where y = any ordinate,
x = the corresponding abscissa,
σ = standard deviation of the distribution,
and π and e have the usual mathematical meanings.

Use of Frequency Curve.—The area of the whole curve is taken as one. In order to find what is the total frequency of the occurrence of a given phenomenon, within a certain range of magnitudes, we have to find the area of the portion of the curve limited by the ordinates at the extremes of that range, the base line, and the curve boundary, e.g., area of portion A B D C in figs. 26. It is convenient to mark the point on the abscissa at which the highest ordinate (or mean) is erected as zero, and to use as the abscissal unit not the group interval or "group interval deviation," x, but the "group interval deviation" divided by the standard deviation, σ, of the distribution, so that the position of any deviation, x, on one or other side of the mean can be expressed as $\pm \frac{x}{\sigma}$ distant from the mean (according as x is situated to the right or left of the mean).

In this way it is possible to express the area of any portion of the curve limited by two ordinates erected at known abscissal points as a decimal fraction of the total area of the curve. This has been done and recorded in what are called *probability integral tables.* The following examples will illustrate the method of working.

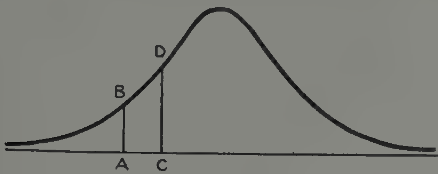

FIG. 26.

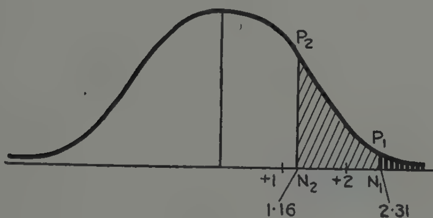

FIG. 27.

(1) The annual number of births in England and Wales is about 750,000, with an average proportion of 51 males to 49 females. How many times in a century may one expect (*a*) more than 383,500 males, (*b*) more than 383,000 males, to be born in one year, assuming that the sex ratio during a series of years follows the normal distribution?

From the formula $\sigma = \sqrt{npq}$ (see p. 70).

$$\text{we get } \sigma = \sqrt{750{,}000 \times 0{\cdot}51 \times 0{\cdot}49}$$
$$= 433 \text{ in round numbers.}$$

The average number of males out of 750,000 births must by hypothesis be $7{,}500 \times 51 = 382{,}500$.

$\therefore$ Abscissal point 383,500 is situated at a distance of 1,000 to the right of the mean ordinate, i.e., $x = 1{,}000$.

$$\therefore \frac{x}{\sigma} = \frac{1{,}000}{433} = 2{\cdot}31 \text{ units to right (point } N_1 \text{ in fig. 27),}$$

and similarly, abscissal point 383,000 is situated

$$\frac{500}{433} = 1{\cdot}16 \text{ units to right (point } N_2 \text{ in fig. 27).}$$

The probability integral table tells us that when $\frac{x}{\sigma} = 2{\cdot}31$, then the portion of the curve beyond it occupies an area of $0{\cdot}0104$ (i.e., about one hundredth of the total area of the curve), and when $\frac{x}{\sigma} = 1{\cdot}16$ the area of the portion of the curve beyond it $= 0{\cdot}1230$ (i.e., about twelve hundredths of the total area of the curve, since the total area of the curve is taken as unity and represents all the various possible frequencies).

Therefore the frequencies 383,500 or more, and 383,000 or more males would occur approximately once and twelve times in a century respectively.

(2) Find at what points along the abscissa the ordinates will enclose half the area of the curve. The probability integral table shows that when the area of the curve enclosed by the median and any ordinate is one quarter of the total area of the curve, then $\pm\frac{x}{\sigma} = 0{\cdot}6745$. Hence, the points required are at a distance on each side of the median equal to $0{\cdot}6745$ of the standard deviation. This explains the reason for the probable error being $= 0{\cdot}6745\sigma$ (see p. 68).

(3) Find the area of the curve from one end to the ordinate erected at the point represented by three times the probable error.

Three times P.E. $= 3 \times 0{\cdot}6745\sigma = 2\sigma$ approximately. The probability integral table shows that the area of the curve between the ordinate erected at this point and its proximal end $= 0{\cdot}02$. From this it is seen that if a measurement $= 3$ P.E., then the probability of its being the true value $= 98 : 2 = 49 : 1$ (see p. 69).

Skew Curves.—We have seen that when the chance or probability of an event happening is equal to that of its not happening, then the frequency of distribution can be represented by a normal frequency curve. There are, however, many natural phenomena in which the probability of an event happening is either greater or less than of its not happening. For example, if instead of taking a *random* sample of men, we were to *select* a number of individuals coming from tall stocks, so that the chances of tall individuals occurring in the sample is greater than that of short individuals, we would find that when the frequency curve is plotted it assumes an asymmetrical shape so that there are more individuals on the tall side of the mean than on the other. Such asymmetrical curves are known as *skew* curves, and their shapes depend on the relative probability of the occurrence and non-occurrence of the event. If, for instance, an event is twice as

likely to happen as not, then the various ordinates in the frequency curve would be represented by the corresponding terms of the expansion $\left(\frac{2}{3}+\frac{1}{3}\right)^n$. If the chances are, say, 5 to 1, then the ordinates would be the terms of the expansion $\left(\frac{5}{6}+\frac{1}{6}\right)^n$, and so on.

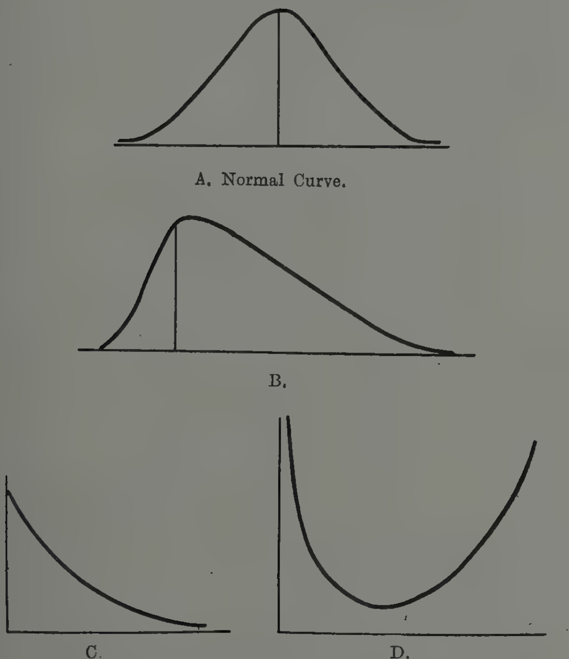

FIG. 28.—Three types of skew curves.

Although the factors responsible for the production of skew curves may be those of pure chance, such curves may also occur in cases where ordinarily the chance of the event happening is the same as that of its not happening, but, as the result of some superimposed removable factor, the balance of chances is disturbed. When, therefore, one finds a skew curve where one would a priori expect a normal frequency curve, one should, as far as possible, endeavour to ascertain what is the nature of the

disturbing factor producing the skewness. For example, if we find that in a large school the heights or weights, or intelligences of the scholars plot out as a skew curve, it would probably be found that there is a preponderance of scholars belonging to a special class, such as race, social status, &c.

Types of Skew Curves.—There are four types of skew curves commonly met with in public health problems. These are shown in fig. 28.

The normal curve fits the following biological statistics: The sex ratios at birth over a series of years for a large community; the heights of persons, including children, either of a mixed population of individuals of all ages or of individuals of the same

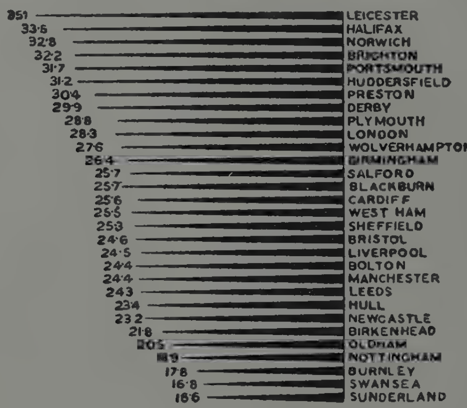

Fig. 29.—Chart showing the improvement (in infant mortality) per cent. in the two means, 1877-1906 and 1907-15, in thirty large towns (S. G. Moore, *The Lancet*, March 14, 1906). The curve is an ogive, and would therefore plot as a normal curve.

age; the lengths of various bones, skull measurements, &c. Mendelian expectations in heredity; birth, marriage, and death-rates in time or space, &c. The normal curve, however, fails to delineate certain other distributions of interest to the student of child hygiene. For example, type B (of the skew curves) fits the age distribution of deaths from measles. Type C would describe the age distribution of infant deaths by months, whilst the U-shaped curve of specific death-rates by ages belongs to type D.

"The frequency curve," says Whipple,[1] "is something more

[1] *Loc. cit.*, pp. 457-459.

than a statistical tool. Properly conceived it stands for a universal principle. Not all the leaves of a tree are alike, not all shells are alike, soldiers are not all of the same height or weight. . . . The frequency curve contains in itself the element of beauty. Moons wax and wane; the tide rises and falls; the flowers of spring come, first a few, then many, and then disappear in the same way, a few lingering into summer. It is said that we live in a world of chance. Nothing is more true. We live in a world where many causes are acting with and against each other. We live in a world of frequency curves. Artists and architects recognise this. The ogee curve is the line of beauty."

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CHAPTER IV

ANTE-NATAL AND INTRA-NATAL MORTALITY

"I saw where in the shroud did lurk
A curious frame of Nature's work.
A floweret crushèd in the bud,
A nameless piece of Babyhood,
Was in a cradle-coffin lying;
Extinct with scarce a sense of dying;
So soon to exchange the imprisoning womb
For darker closets of the tomb!

. . . .

Riddle of destiny, who can show
What thy short visit meant; or know
What thy errand here below?
Shall we say that Nature blind
Checked her hand and changed her mind,
Just when she had exactly wrought
A finished pattern without fault?"

CHARLES LAMB

IN the absence of State registration of pregnancy, abortion, or stillbirth, it is impossible to know exactly what proportion of pregnancies terminate prematurely with the death of the fœtus. Various estimates have been made, but we may, I think, provisionally accept the figures given by Ballantyne, viz., 15 per cent. of all conceptions. In other words, the total births represent no more than 85 per cent. of the total conceptions.

Now, available statistics go to show that for every 1,000 live births there are about thirty-five stillbirths, so that we can say that every 1,035 births (alive or dead) represent $\frac{100}{85} \times 1{,}035 = 1{,}218$ conceptions. In other words, starting with 1,218 conceptions, we only get 1,000 live births, so that for 1,000 infants born alive at least 218 fœtal lives have been sacrificed. We see, therefore, that the fœtal mortality rate is not less than 218 per 1,000 live births. Taking 750,000 as the approximate annual number of live births in England and Wales, we arrive at the fact that no less than $218 \times 750 = 163{,}500$ fœtal lives are lost in this country every year. According to Karl Pearson, the total

ante-natal mortality is 605 out of every 1,000 born alive at full term.[1] But this is not all, for many of the infants that die during the early weeks of infancy owe their deaths to conditions connected with ante- and intra-natal life. The figures given by the Registrar-General show that in 1923 there were nineteen deaths (per 1,000 born) during the first post-natal month—due to prematurity or fœtal malnutrition—so that the number of premature deaths during that year were $19 \times 750 = 14,250$. Hence, the total actual fœtal mortality may be put down as $163,500 + 14,250 = 177,750$ per annum in England and Wales.

All this calculation is, however, merely an estimate, and in the absence of compulsory notification of abortions and miscarriages, the magnitude of the loss can never be accurately known. Again, without such compulsory notification it is impossible to come to any very precise knowledge of the various causes of ante-natal death. If every such mishap were notifiable and the specimen forwarded to a State laboratory for expert anatomical, microscopic and biochemical examination, much useful information would be acquired regarding the various causes of ante-natal death and the manner of preventing them. Considerable numbers of fœtuses (561 in all), forming fair random samples of the hospital class, have been examined by Eardley Holland (300) and F. J. Browne (261), and their findings —which are in remarkable agreement—form most valuable contributions to the subject, but the numbers are not sufficiently large, nor are they quite representative of all the various parts of the country or of the various strata of society.[2] A consideration, however, of the manner in which the fœtus lives and derives its nourishment will throw a good deal of light upon the manner in which it ceases to live. We shall deal with the subject of fœtal nutrition in some detail later (see Chapter VIII), but we shall give a very brief outline of the subject here in order that the reader may be able to understand what are the various factors which may—from a theoretical standpoint—have an influence upon the life and health of the fœtus.

Physiological Connection between Mother and Fœtus.—At a very early stage of embryonic development, a close and intimate

1 "The Chances of Death," London, 1897, vol. i, pp. 24-39.

2 Since the above was written the Medical Research Council have published a Report on Dead Births and Neo-natal Deaths based on 1,673 cases investigated by different workers in different parts of the country (Special Report Series, No. 109).

connection is established between the mother and the future infant by means of the placenta, which is a structure consisting of a large number of finger-like processes or tufts, called chorionic villi (coming from a portion of the membranes surrounding the embryo), richly supplied with blood-vessels, interdigitating with corresponding hollows or vascular spaces called sinuses, in the wall of the uterus or womb. In this way the blood of the growing unborn infant is brought into close approximation with that of the mother, so that not only does food material and oxygen pass from the mother to the fœtus and excretory products or waste substances from the fœtus to the mother, but also substances not normally found in the blood may, if abormally present in the maternal circulation, pass from the mother to the fœtus. Such substances are poisons, toxins, microbes, drugs, &c. Moreover, experiment has shown that of the nutritive substances some pass through the placenta from mother to fœtus by a physical process of diffusion, so that the more there is of those substances in the maternal blood the more of them will pass to the fœtus, and vice versa; whilst other substances pass from the mother to the fœtus by a process of vital selective action on the part of the placenta, so that whether the maternal blood contains much or little of them the fœtus takes as much of these substances as it requires—neither more nor less. In other words, in the case of the one group of substances, like sugar, the fœtus takes as much as the mother can give it, whilst in the other groups of substances, like fats and mineral salts, and possibly proteins, the fœtus takes as much as it requires—whether the mother can afford them or not.

With these facts in our minds, and remembering that the fœtus results from the fusion of the male and female sexual cells, we can now classify the **theoretical causes of fœtal malnutrition or death** as follows :—

ANTE-NATAL CAUSES.

A. **Inherent defects in ovum or spermatozoon**, resulting either in sterility or in early post-conceptional death. Arthur Robinson believes this unavoidable pre-natal mortality to be responsible for 40 per cent. of the total pre-natal mortality in human beings (see p. 11).

B. **Quantitative nutritional deficiency**, or **defective supply of normal blood**, such as occurs in

(a) *Absolute placental deficiency*, viz., an abnormally small placenta. The average normal weight of a placenta is

about one-eighth of that of the fœtus. If it is less than one-tenth of the fœtal weight it may be considered abnormally small. Eardley Holland found this to be the case in a considerable number of dead fœtuses where no other cause for the death could be discovered.

(*b*) *Relative placental deficiency* due to

(1) PREMATURE SEPARATION OF THE PLACENTA.

(α) When normally situated, as in accidental hæmorrhage (profuse bleeding), or in retro-placental hæmatoma (slight bleeding). Both these conditions may be due to

(i) Toxæmia resulting in erosion of the blood-vessels; or (ii) injury such as blows on the abdomen, or falls during pregnancy.

(β) When abnormally situated—as in placenta prævia.

(2) PLACENTAL DISEASE OR ABNORMALITY, e.g., obliteration of the lumen of the blood-vessels of the villi due to syphilis, or absence of one or more arteries carrying blood from mother to fœtus.

(3) ANY MATERNAL DISEASE RESULTING IN DEFECTIVE CIRCULATION THROUGH THE PLACENTA, e.g., heart disease, or tuberculosis, &c.

(4) MULTIPLE PREGNANCY, such as twins, triplets, &c., when the placenta is not sufficient in size to provide for the needs of each fœtus.

(*c*) Obstruction to placental circulation, e.g., knots or twists in the cord, or compression of cord during birth.

C. **Qualitative nutritional deficiency**, or a **normal supply of defective blood**, such as occurs in syphilis, pregnancy toxæmia (especially eclampsia and albuminuria), alcoholism, infectious diseases (influenza, &c.), diabetes, poisoning by lead, phosphorus, arsenic, &c. These toxins or poisons may either injure the placenta and bring about premature labour, or pass through the placenta and kill the fœtus.

D. **Excessive malnutrition or overwork of the mother** (see p. 127 *et seq.*).

E. **Injury during pregnancy**, such as blows or falls, possibly resulting in separation of the placenta.

II. **INTRA-NATAL** or **OBSTETRIC CAUSES** of stillbirth, e.g., injuries received by fœtus at birth as the result of faulty technique in breech or transverse presentations, or of instrumental delivery.

III. **Deliberate induction of abortion** — either medically or criminally.

IV. **Malformation**, i.e., anencephaly, hydrocephaly, &c.

V. **Unknown causes.**

The above theoretical classification of causes of fœtal death simplifies itself into the following (Eardley Holland) :—

(1) Those the causes of which can be discovered and prevented (or treated) during the ante-natal period :—

- (*a*) Syphilis.
- (*b*) Toxæmia of pregnancy.
- (*c*) Malpresentations, e.g., breech or transverse—which can be rectified by external version, or occipito-posterior positions.

(2) Those the cause of which can be discovered ante-natally, but which depend for their prevention on special intra-natal treatment :—

- (*a*) Contracted pelvis—for which induction of premature labour or Cæsarean section, &c., may be adopted. To this may be added post-maturity of the fœtus (over nine months' gestation with its consequent large size) Christine J. Thomson found this to have been a cause of intra-natal death in 3 per cent. of her series of cases.[1]
- (*b*) Cases of toxæmia of pregnancy which are not amenable to ante-natal treatment and for which induction of premature labour may be necessary.

(3) Those the causes of which can only be discovered and treated during the intra-natal period :—

- (*a*) Ante-partum hæmorrhage.
- (*b*) Prolapse of cord.

(4) Those the causes of which can for the present not be discovered or treated at any time :—

- (*a*) Severe fœtal malformations.
- (*b*) Placental insufficiency (absolute).
- (*c*) Umbilical knots.

[1] *Journ. Obst. and Gynæc. Brit. Emp.*, 1926, vol. xxxiii, p. 415.

F. J. Browne[1] gives the following table of causes of fœtal deaths (in one of his series of 127 cases):—

TABLE II.

I. Stillbirths.	
(1) **Toxæmic.**	
(a) Syphilis (in 15 spirochætes were found in the fœtal organs)	24
(b) Placental infarction	10
(c) Utero-placental apoplexy	4
(d) Premature senility of placenta	1
(e) Maternal glycosuria	1
(f) Eclamptic convulsions	6
(g) Drugs	—
(2) **Mechanical.**	
(a) Placenta prævia	2
(b) Traumatic separation of placenta	—
(c) Knots of umbilical cord	1
(3) **Developmental.**	
(a) One artery instead of two in cord (?)	1
(b) Small placental area	1
(c) General fœtal œdema	1
(d) Other congenital anomalies	—
(4) **Undetermined**	8
	60
II. Intra-natal Deaths.	
(1) **Traumatic.**	
(a) Craniotomy	11
(b) Cerebral hæmorrhage	14
(c) Asphyxia	36
(d) Suprarenal hæmorrhage	1
(e) Other injuries	—
(2) **Infective.**	
Pneumonia (present also in four other cases included under craniotomy)	1
(3) **Toxæmic.**	
Scopolo-morphine narcosis	—
(4) **Developmental**	4
(5) **Undetermined**	—
	67

Hence asphyxia is by far the commonest cause (more than 50 per cent.); cerebral hæmorrhage comes next.

Whitridge Williams, writing on "The Significance of Syphilis in Pre-natal Care and in Causation of Fœtal Deaths,"[2] gives the following table of fœtal deaths:—

[1] *Edin. Med. Journ.* (Obst.) September, 1924.

[2] *Bulletin Johns Hopkins Hospital*, xxxi, No. 351, May, 1920, p. 143.

TABLE III.

Cause of death	Number of cases	Percentage
Syphilis	104	34·44
Dystocia	46	15·20
Toxæmia	35	11·55
Prematurity	32	10·59
Cause unknown	26	8·61
Separation of placenta ..	16	*5·28
Deformity	11	3 64
Eleven other cases	32	10·69
	302	100

Beckwith Whitehouse, who analysed the results at the Birmingham Maternity Hospital during the period 1900-19, finds that out of a total of 715 fœtal deaths there were (exclusive of syphilis, which were not admitted) :—

TABLE IV.

Constitutional maternal disease (heart, &c.)..	18 =	2·5	per cent.
Toxæmia of pregnancy	94 =	13·1	,,
Placental causes	256 =	35·8	,,
Obstruction to placental circulation (prolapse of cord, &c.)	27 =	3·8	,,
Obstructed labour..	192 =	26·8	,,
Fœtal malformation, &c.	36 =	5·0	,,
Prematurity	30 =	4·2	,,
Unknown	62 =	8·6	,,
	715	99·8	

The following statistical table of causes of fœtal death is given by Eardley Holland :—

TABLE V.

Ante-natal—		
Syphilis	16 per cent.	49 per cent.
Pregnancy toxæmia	10 ,,	
Unknown causes	10 ,,	
Placental causes (hæmorrhage, insufficiency, &c.)	6 ,,	
Fœtal deformity	5 ,,	
Maternal	2 ,,	
Intra-natal (complications of labour)		51 per cent.

In the recent compilation, by Eardley Holland and Janet Lane-Claypon, for the Medical Research Council (Special Report Series, No. 109, published in 1926), the incidence of the various causes of fœtal death in different localities varied as follows :—

TABLE VA.

Cause of death	Minimum incidence	Maximum incidence
Complications of labour..	30·2 per cent. (Cardiff)	40·2 per cent. (Glasgow)
Ante-partum hæmorrhage	10·5 ,, ,, (Edinburgh)	30·7 ,, ,, (Glasgow)
Toxæmia of pregnancy ..	7·7 ,, ,, (Liverpool)	20·1 ,, ,, (Cardiff)
Syphilis	7·0 ,, ,, (Glasgow)	11·1 ,, ,, (Edinburgh)
Maternal states	0·8 ,, ,, (Cardiff)	5·9 ,, ,, (London)
Placental states	0·0 ,, ,, (Glasgow)	2·0 ,, ,, (Liverpool)
Fœtal states	5·0 ,, ,, (London)	23·6 ,, ,, (Liverpool)
Prematurity	0·0 ,, ,, (London)	10·4 ,, ,, (Edinburgh)
Cause unknown	3·2 ,, ,, (Glasgow)	11·3 ,, ,, (London)

It will thus be seen that the ranges of variation in the incidence of the various causes of dead births in different localities are very considerable. All the results agree, however, that complications of labour are the most common cause of fœtal death.

In what follows, we shall deal with those causes of fœtal death which are controllable. It is clear, for instance, that placental deficiency is a cause which we cannot prevent. The same of course applies to inherent defects in the ovum or spermatozoon, as well as to fœtal deformity. On the other hand, some 60 per cent. of the intra-natal causes, the whole of the mortality due to syphilis, and some 60 per cent. of the fœtal deaths resulting from pregnancy toxæmia, are preventable, and should be prevented by proper ante-natal supervision and treatment.

In dealing with statistics of ante-natal deaths we must distinguish between abortion and miscarriages, and stillbirths.

Abortion or **miscarriage** means the expulsion of a dead fœtus before it is viable, i.e., before the twenty-eighth week of pregnancy. By a **stillbirth**, as we understand it in England, is meant the birth of a dead fœtus at a period at which it is viable, i.e., after the twenty-eighth week of pregnancy. If the child has breathed after birth, it is not a stillbirth. Abortions or miscarriages are neither registrable nor notifiable. A stillbirth is notifiable under the Notification of Births Act, 1907 (i.e., the doctor or midwife present at the birth, or the nearest relative, must notify the event to the local Medical Officer of Health), but is not registrable as is the case with live births.

A stillbirth may occur as the result of the death of the fœtus (*a*) during the ante-natal period, or (*b*) shortly before or during birth.

When the fœtus dies long before birth its body is born in a macerated condition. If the fœtus dies shortly before or

during labour, its body is in a fresh condition. The proportion between macerated and fresh bodies amongst stillbirths is approximately 2 : 3.

The Measurement of Ante-Natal Mortality.—When the mortality of the general population is studied it is usual to state a death-rate as the number of deaths per year per 1,000 total population during that year. In the study of problems relating to special groups or classes of population, such as of people of a given age-group, or of a given sex or race, &c., one employs a *specific death-rate*, by which is understood the annual number of deaths of people in that group per 1,000 persons in the same group during the same year. The calculation of fœtal mortality should therefore be logically based on the specific death-rate of the conceptional age-group from the moment of conception to the end of pregnancy, and its most correct expression would be the annual number of fœtal deaths per 1,000 conceptions occurring during the year. Such a mortality rate would not only give one an exact idea of the magnitude of the problem, but would enable one to compare the fœtal mortalities of different countries as well as in the same country at different times. This method of estimating fœtal mortality rates is, however, for the present impossible, because not only are abortions, miscarriages and stillbirths not registrable, but even if their absolute number per annum were known we would still require to know what has been the total number of conceptions during the year, and this could only be accurately ascertained by a complete system of registration of pregnancy—an ideal of perfection at which we may aim but which for many reasons is at present unattainable. It might be thought that registration of abortions, miscarriages and stillbirths would, together with the perfect system of registration of live births, give us complete information of the annual number of conceptions, but although this would give us a very approximate idea it would not give us all the information, since the death of an expectant mother from some cause unconnected with pregnancy would fail to be recorded as a "conception." For these reasons the most convenient way of expressing a fœtal mortality rate is "per 1,000 live births," since the annual number of live births is, in this country at any rate, known with the utmost accuracy. But even with such a denominator, we cannot estimate correctly the extent of the total fœtal mortality because, as I have already stated, abortions or stillbirths are in this country not registrable, although the latter are notifiable

under the Midwives Act. The following tables, from Newsholme[1] and Pearl[2] respectively, give estimates of the number of *still-births* per 100 live births in different areas of England and the United States at different years :—

TABLE VI.—SHOWING PERCENTAGE OF STILLBIRTHS (PER 100 LIVE BIRTHS) IN VARIOUS AREAS BETWEEN 1910 AND 1912 (Newsholme).

Areas	1910	1911	1912	Range of percentage for three years, 1910-12
In 74 county boroughs and towns with over 50,000 population	3·01	3·18	2·98	1·08 - 5·49
In 67 towns with populations between 20,000 and 50,000	3·21	3·07	3·34	1·19 - 7·82
In the 29 Metropolitan boroughs ..	2·14	2·21	2·46	0·31 - 5·83

TABLE VII.—BIRTHS, STILLBIRTHS, STILLBIRTHS PER 100 LIVE BIRTHS AND AVERAGE NUMBER OF CHILDREN EVER BORN, FOR WHITE CHILDREN BY COUNTRY OF BIRTH OF MOTHER, 1918 (Raymond Pearl).

Country of birth of mother	Births	Still-births	Still-births per 100 live births	Average number of children *ever* born
United States	838,000	30,891	3·7	3·1
Total Foreign	314,780	12,392	3·9	3·9
Canada	13,861	651	4·7	3·5
Ireland	14,038	620	4·4	3·3
England, Scotland and Wales	11,069	473	4·3	3·3
Italy	71,373	2,989	4·2	4·4
Russia (including Russian Poland) ..	60,269	2,369	3·9	3·3
Austria (including Austrian Poland) ..	62,111	2,384	3·8	3·9
Other foreign countries	22,355	835	3·7	3·6
Denmark, Norway and Sweden ..	11,624	413	3·6	3·6
Germany (including German Poland)..	15,414	559	3·6	4-6
Poland (not specified)	14,534	525	3·6	4·2
Hungary	18,132	574	3·2	3·8

[1] "Vital Statistics," London, 1923, p. 81.

[2] "The Vitality of the Peoples of America," *Amer. Journ. Hyg.*, i, 1921, pp. 592-674.

From Table VII it is seen that high fertility in the past is generally associated with a low stillbirth-rate. In other words, the more children a woman has had the less likely is her next birth to be a stillbirth.

These figures, however, cannot by any means be considered as absolutely reliable—although they are probably approximately correct. The errors attending these statistics are the following. In the first instance, it is impossible to determine with any degree of exactness the true period of gestation, so that we cannot say whether the infant would have been viable. Secondly it is quite certain that many stillbirths fail to be notified. That this is so is proved by the discrepancy between the number of live births that are registered and those that are notified, and if some live births escape notification, it is certain that the leakage amongst stillborn babies must be greater still. But apart from these errors, there are differences in the definition of stillbirths in different countries which make comparison of stillbirth-rates between one country and another impossible. Thus in France, not only are infants considered stillborn if they are born without sign of life, but also those born alive but which die before registration (i.e. within three days after birth) are included under the same term, and in Switzerland a stillbirth is the birth of a dead fœtus after six months' pregnancy. In New York State a stillbirth is the birth of a dead fœtus after five months of gestation. Again, it is not possible to say at what age a fœtus is viable. The Committee of the Royal Statistical Society (1912) reported:—

(1) That it is desirable that an international definition of stillbirths should be adopted.

(2) That stillbirths should be registered.

Stillbirths are registrable in a number of countries like Austria, Belgium, Bulgaria, Germany, New Zealand, Norway, Sweden, Switzerland, the United States, &c., although the definition of a stillbirth is, as has already been mentioned, not the same in each country.

The following Table VIII, given by Fuller,[1] gives the stillbirth-rate (of infants born dead after the sixth month of pregnancy) for the various Swiss cantons:—

[1] Edward Fuller, *The International Year Book of Child Care and Protection*, 1925, p. 461.

TABLE VIII.

Swiss canton	Infantile mortality per 1,000 live births	Total stillbirths per 1,000 live births	Illegitimate stillbirths per 1,000 live births
Aargau..	68	28	102
Appenzell, Intérieur..	73	39	—
,, Extérieur..	123	42	—
Bâle Province.. ..	50	25	58
,, Town	40	75	67
Bern	62	34	57
Fribourg	113	25	21
Geneva	92	30	—
Glarus	74	41	—
Grisons	89	23	56
Lucerne	78	26	58
Neuchâtel	71	34	26
Nidwalden	88	11	—
Obwalden	87	11	—
St. Gall	74	26	61
Schaffhausen	68	21	60
Schwyz	74	21	34
Soleure	72	25	71
Thurgau	64	34	63
Ticino	121	28	58
Uri	101	23	71
Valais	117	16	17
Vaud	66	33	34
Zug	77	25	—
Zürich	58	35	57

TABLE IX.—STATISTICS REGARDING STILLBIRTHS AND ABORTIONS.[1]

	Percentage to live births	
	Still-births	Abortions
Philadelphia (La Fetra) ..	5·0	20·0
England and Wales (1908-1912) –		
London	2·18	..
74 large towns and boroughs..	3·06	..
67 small towns	3·34	..
Boston, U.S.A.	3·9	..
Manhattan, U.S.A.	4·8	..
Dr. Priestley's statistics ..	..	23 per cent. of all pregnancies 30 per cent. of all live births
Dr. Whitehead's statistics ..	..	14 per cent. of all pregnancies 16.6 per cent. of all live births
Dr. Whitridge Williams's statistics	..	16·20 per cent. of all pregnancies
Dr. Malins, of Birmingham ..	..	19·23 per cent. of all pregnancies
Professor Taussig	..	30 per cent. of all pregnancies
Dr. Amand Routh	2·8	27·4 per cent. of all pregnancies

[1] Amand Routh, "Ante-natal Hygiene," *Brit. Med. Journ.*, February 14, 1914, p. 356.

Amand Routh has estimated that there are four abortions and miscarriages to every stillbirth. If therefore we take 3·5 per cent. as the stillbirth-rate, we get the abortion and miscarriage rate as 14 per cent., and the total ante-natal mortality rate, other than those that occur in the germinal stage, as 17·5 per cent. of all live births, which is approximately the same as the estimate given by Ballantyne.

From the above statistics it is seen that the stillbirth-rate is higher for illegitimate than for legitimate children. This, which is also the case with abortion, &c., holds good also, as we shall see, for post-natal infantile mortality, and is probably due to the deliberate induction of premature birth in the case of illegitimate pregnancies. That it is not due to any influence of maternal environmental conditions is shown by the statistics quoted later, on page 123 *et seq.*, to the effect that economic conditions have no appreciable influence on the stillbirth-rate. It is further shown by the fact that there is no seasonal variation in the stillbirth-rate, as there is in the case of infant mortality rate. The variation in stillbirth-rates in different countries is not due to any geographical or climatic causes, but can be entirely explained by the imperfect registration and variation of definition of stillbirths. It is therefore apparent rather than real.

Syphilis.—The syphilitic agent may affect the future child in a number of ways and at different ante-natal stages. It may infect the ovum during conception through the paternal semen. At a later stage it may either cause relative placental deficiency by obliterating the lumen of the placental blood-vessels, or it may pass through the placental circulation and infect the fœtus. It is probable, however, that infection of the mother after the seventh month of pregnancy fails to infect the fœtus, because the placenta is then so strongly developed that it manufactures a special protective ferment preventing the passage of the syphilitic organism from mother to fœtus. Syphilis used to be considered the most important cause of fœtal death, but statistics show that this is not so if we include *intra*-natal deaths under the term fœtal death. It is, however, undoubtedly the most important cause of *ante*-natal deaths alone, it being responsible for some 30 to 40 per cent. of ante-natal mortality. But taking 20 per cent. as being approximately the proportion of *total* fœtal deaths due to syphilis, we get the following calculation: The total annual number of ante-natal deaths in England and Wales is, as we have seen on page 97, about 178,000. Taking 20 per cent. of this, we obtain

approximately 35,600 fœtal deaths that are caused by syphilis every year in this country. Fortunately this disease is one which can be both diagnosed and effectively treated ante-natally (see Table XII and p. 110).

TABLE X.

Abortions and Premature Births.

	Syphilitic	Non-syphilitic	Authority
Abortions during the first three months	31·8 per cent.	30·6 per cent.	Adair
Abortions during the second three months	30·4 ,,	30·7 ,,	
Total abortions during the first six months	6·17 ,, (6 out of 94 cases)	13·7 ,,	Cruickshank
Premature births	32·54 per cent.	19·88 ,,	
Stillbirths amongst prematures	68·75 ,,	34·61 ,,	

TABLE XI.

Incidence of Syphilis in Pregnant Women.

Expectant mothers	Percentage of syphilis
In all pregnant women	6 - 9
In mothers of premature infants	30 - 32·5
In mothers of stillborn infants	10

TABLE XII.

Results of Ante-natal Treatment of Syphilis.

	Untreated	Treated	
		Satisfactorily	Inefficiently
Stillbirths among syphilitic women	60·6 per cent. (Ballantyne)	5·1 per cent. (Ballantyne)	—
	52 per cent. (Williams)	7·9 per cent. (Williams)	37 per cent. (Williams)

The general belief that syphilis is a predominating cause of early abortion has not been borne out by recent work. It has been shown [1] that the percentage of abortion before the end of

[1] Cruickshank, J. N., "Maternal Syphilis as a Cause of Death of the Fœtus and of the Newborn Child," Med. Res. Council, Special Report Series, No. 82.

the sixth month is not more amongst syphilitic than amongst non-syphilitic women. On the other hand, syphilis is one of the most important causes of death of the fœtus with its premature expulsion in the later months of pregnancy. (See Tables X and XI.)

The statistics show that :—

(1) The incidence of syphilis in pregnant women is 9 per cent.

(2) The incidence of early abortion is the same in syphilitic as in non-syphilitic cases, but that death of the fœtus in later months is about twice as common in syphilitic cases.

(3) Congenital syphilis is by no means so common as it was believed. It is only present in 1 per cent. of all births.

(4) A positive Wassermann reaction in a new-born infant is no proof of its being syphilitic. It may be due entirely to the presence within its blood of reacting substances that passed through the placental circulation (Cruickshank).

(5) The most satisfactory results can be obtained by means of ante-natal treatment of syphilis.

The following figures give the ante-natal and post-natal deaths due to syphilis in France :—

Abortions	40,000	per annum.
Stillbirths	20,000	,,
Deaths at all ages	80,000	,,
	140,000	

The following figures of fœtal mortality are from the Clinique Baudelocque in Paris :—

Syphilis contracted during pregnancy :—			
Not treated	70	per cent.	mortality.
Treated (during last 3 months of pregnancy)	30	,,	,,
Syphilis contracted before gestation :—			
Not treated	65	,,	,,
Treated (during last 3 months of pregnancy)	5	,,	,,
Old syphilis :—			
Not treated	20	,,	,,
Treated	8	,,	,,

The Wassermann Reaction in Pregnancy (F. J. Browne) :—

(1) A strongly positive Wassermann reaction in a pregnant woman is very decisive of syphilis.

(2) The fœtus of such a woman need not, however, be syphilitic.

(3) A woman with a negative Wassermann reaction may successively bear syphilitic fœtuses (although none of these fœtuses will have spirochætes in their organs).

(4) The mother of a spirochæte-positive fœtus invariably has a positive Wassermann reaction.

(5) On the strength of 100 cases, Browne is of opinion that pregnancy does not modify the Wassermann reaction. Thus, no case of positive pre-pregnancy Wassermann became negative during pregnancy (apart from treatment). Nor has a negative Wassermann reaction during pregnancy become positive post partum.

The Effect of Syphilis upon the Spermatozoa.—V. Widakowich[1] found that :—

(1) Out of forty-one cases of known syphilis, all showed the presence of 5·1 per cent. to 7·78 per cent. abnormal spermatozoa (viz., those with 2, 3 or 4 heads, or 2, 3 or 4 tails; giant and dwarf forms; micro- and macrocephalic, &c.).

(2) Out of thirty-five cases with suspected syphilis, seventeen showed a positive Wassermann reaction as well as many (5·48 per cent.) abnormal spermatozoa; nine cases showed negative Wassermann reaction and few abnormal spermatozoa; nine cases showed negative Wassermann reaction with many abnormal spermatozoa.

(3) Twenty-four healthy individuals had 1·84 per cent. abnormal spermatozoa. Of these eight were married and had normal children.

Alcohol.

Whilst there are no two opinions with regard to the harmful effects of syphilis upon the life and welfare of the fœtus, authorities are not in entire agreement with respect to the direct influence of alcohol on ante-natal life—as contrasted with the indirect environmental influences accompanying alcoholism, such as neglect, &c., which nobody disputes.

(*a*) **Influence of Parental Alcoholism upon Offspring.**—We shall deal with the question of alcohol in relation to ante-natal life later (see p. 243); here we shall give a few statistics based upon experiments on animals as well as observations in man. We see that the human statistics—which are probably very reliable—do not agree with those obtained from experiments upon animals with regard to the influence of parental alcoholism upon the offspring. The difference is due to the comparatively much higher dose of alcohol to which experimental animals are exposed. Experimental and pathological evidence, however, leaves no doubt that alcohol can and does pass from the maternal into the fœtal circulation, *and if transferred in large quantities*, as is the case with chronic excessive drinkers, the alcohol has a prejudicial effect upon the fœtus.

[1] *La Semana Medica*, Buenos Aires, 1920, xxvii, p. 633.

TABLE XIII.—EXPERIMENTAL RESULTS OF EFFECTS OF PARENTAL ALCOHOLISM ON OFFSPRING.

Stockard's Experiments of Alternate Matings of Normal Guinea-pigs with Normal and Alcoholic Mates.

	Matings of 35 normal males successively with		Matings of 44 normal females successively with	
	Normal females	Treated females	Normal males	Treated males
Number of matings	81	81	77	81
Total offspring	196 (average litter, 2·42)	185 (average litter, 2·28)	195 (average litter, 2·53)	182 (average litter, 2·25)
Failure to conceive	6 (= 7·9 %)	6 (= 7·9 %)	3 (= 3·89 %)	10 (= 12·34 %)
Lived over 3 months	151 (= 77·03 ,,)	105 (= 56·64 ,,)	161 (= 82·56 ,,)	118 (= 61·83 ,,)
Died under 3 months	45 (= 22·96 ,,)	80 (= 43·35 ,,)	34 (= 17·45 ,,)	64 (= 35·16 ,,)
Defective ..	0	11 (= 5·95 ,,)	0	9 (= 4·97 ,,)

This table shows that alcoholic males are more sterile than normal ones; that the offspring of alcoholic fathers or mothers have a higher early post-natal mortality; and that defective offspring are common in the case of alcoholic parents.

TABLE XIV.

Laitinen's Experiments on Weights at Birth, and the Development of Guinea-pigs during the first 60 *days.*

	Birth weight in grm.	Days					
		10	20	30	40	50	60
Control animals	69·30	107·85	158·8	208·2	237·74	280·73	317·56
Mothers only alcoholic	61·27	92·18	126·58	172·06	202·64	247·08	282·97
Fathers only alcoholic	61·18	95·79	130·00	184·71	228·82	268·76	294·21
Both parents alcoholic	54·88	100·94	146·14	197·5	238·74	270·33	290·17

TABLE XV.—PEARL'S EXPERIMENTS WITH CHICKS.

Nature of ancestry of chicks	Number of matings	Total eggs set	Total zygotes formed	Zygotes formed (per cent.)	Pre-natal mortality (per cent.) in round numbers	Post-natal (180-day) mortality per cent.	Zygotes alive at 180 days (per cent.)
Both parents alcoholic ..	5	82	18	22	11	25	67
Dam only alcoholic ..	3	63	5	8	80	0	20
Sire only alcoholic ..	13	512	274	54	47	14	46
Sire and one or more grandparents alcoholic	3	233	158	68	47	28	34
One or more grandparents alcoholic	3	187	113	60	46	7	50
All of alcoholic ancestry ..	27	1,077	568	53	46	17	45
All of non-alcoholic ancestry	28	1.333	1,060	80	56	21	35
Differences between alcoholic and non-alcoholic	– 1	– 256	– 492	– 27	– 10	– 4	+ 10

The table brings out the following facts:—

(1) Every 100 eggs from birds of the alcoholic series produced only fifty-three embryos as compared with eighty embryos produced by 100 eggs from the non-alcoholic series (i.e., 27 per cent. less). Effect is mostly on germ cells of female.

(2) Of the embryos formed, the pre-natal mortality was 10 per cent. less in the alcoholic series than in the non-alcoholic (46 per cent. and 56 per cent. respectively).

(3) The early post-natal mortality was also lower by 4 per cent. in the alcoholic series (17 per cent. as against 21 per cent.).

TABLE XVI.—STATISTICAL RESULTS RELATING TO INFLUENCE OF PARENTAL ALCOHOLISM ON OFFSPRING IN MAN (Elderton).

	Condition of parents in relation to alcohol	Mean height (in.)	Mean weight (lb.)	Mean age (yrs.)	Correlation coefficients: Drink and height	Correlation coefficients: Drink and weight	Correlation coefficients: Drink and greater age	Partial correlation coefficients: Drink and height (constant age)	Partial correlation coefficients: Drink and weight (constant age)
Sons	Sober Drink	47·5 47·9	53·8 55·0	9·4 9·8	– 0·7	– 0·06	0·11	0·04 ± 0·03	0·05 ± 0·03
Daughters	Sober Drink	46·8 46·6	52·7 52·3	9·3 9·4	0·02	0·02	0·03	0·09 ± 0·03	0·08 ± 0·03

TABLE XVII.

		Correlation coefficients					Partial correlation coefficients	
		Drink and height	Drink and weight	Drink and greater age	Age and height	Age and weight	Drink and height (age constant)	Drink and weight (age constant)
Sons	Father	− 0·04	− 0·05	0·08	0·81	0·81	0·04 ± 0·03	0·04 ± 0·03
	Mother	− 0·01	− 0·01	0·07	0·81	0·81	0·08 ± 0·03	0·08 ± 0·03
Daughters	Father	0·03	0·04	0·00	0·81	0·82	0·03 ± 0·03	0·04 ± 0·03
	Mother	0·02	0·03	0·07	0·81	0·82	0·13 ± 0·03	0·14 ± 0·03

The only really significant (though very slight) correlation is between **maternal** alcoholism and physique of daughters which suggests **environment rather than direct toxic influence.** (r = 0·13 ± 0·03 and 0·17 ± 0·03 ∴ r > 3 P.E.).

(1) More alcoholic mothers go out to work than non-alcoholic (43·6 per cent. : 26·4 per cent.).

(2) More domestic work thrown on daughters than on sons.

TABLE XVIII.

	Father			Mother		
	Sober	Drinks	Bouts	Sober	Drinks	Bouts
Average number of dead children	1·73	1·99	1·97	1·68	2·09	2·28
Average size of family ..	5·99	6·20	6·03	5·91	6·25	6·32
Nett family	4·26	4·21	4·06	4·23	4·16	4·04
Death-rate per cent. ..	28·9	32·1	32·7	28·4	33·4	36·1

r (**Father** drinking and number of dead children) = 0·06 ; r (father drinking and size of family) = 0·04.

r (**Mother** drinking and number of dead children) = 0·12 ; r (mother drinking and size of family) = 0·01.

∴ Drinking of mother is associated with higher child mortality. **This is mostly due to accidents,** such as overlaying, &c., since highest death-rate occurs when mother drinks in bouts.

(*b*) **Influence of State of Inebriety during Conception upon Offspring.**—The belief in evil effects of alcoholism upon conception dates from very early times. The ancient Greeks represented the deformed Vulcan as having been begotten by Jupiter inebriated with nectar, and Diogenes, on seeing a stupid child

inferred that its father was drunk when its mother conceived it. Similar opinions were held by the Talmudic Rabbis, who believed not only that the children of sober parents were stronger and more beautiful than those of drunkards, but that conceptions occurring during states of inebriety resulted in mentally deficient offspring. In other words, it was believed that germ cells impregnated with alcohol gave rise to defective individuals. Of recent years the subject has been investigated statistically by Bezzola and Schweighofer, who presented figures to show that the products of conceptions occurring during times of inebriety, such as times of carnival and vintage, resulted in a large percentage of stillbirths and idiocy. But Pearson and Elderton have definitely shown that Bezzola's statistics are mathematically unsound, because they found that the differences in the numbers of mental defectives between the two groups of offspring, as found by Bezzola, were not more than could be accounted for by the theory of pure chance, since they fall well within the probable error limit (see "Eugenic Laboratory Memoirs," Nos. X and XIII).

(*c*) **Influence of Alcoholism in Expectant Mother upon the Embryo and Fœtus.**—The embryonic stage is the stage when the various organs and limbs are formed, and any noticeable effect of alcohol would be an excessive production of monstrosities and early abortions. Here again there are no reliable statistics upon which to form an opinion, although the experiments of Pearl and Stockard (already quoted) show that in the case of hens' eggs, or of guinea-pigs receiving alcohol in early stages of development, there is an excessive proportion of malformed offspring. Experiments on pregnant animals, as well as on human beings (Nicloux), have shown that alcohol taken by the mother can be detected in the blood of the fœtus. It has, moreover, been shown that within an hour and a half there is established osmotic equilibrium for alcohol between the maternal and fœtal bloods (see table of Dr. Kostitch's experiments, Chapter VIII). As the fœtal tissues are very delicate and in a state of rapid growth, alcohol can only have a deleterious effect upon them. Until we have notification of abortions we shall never know the extent of wastage of fœtal life resulting from alcohol. But Sullivan found that confirmed women drinkers who continued to drink during pregnancy had large proportions of premature and stillbirths, and when the supply of drink was compulsorily stopped in the second half of pregnancy, *these same women* gave birth to living infants.

(*d*) **Intra-natal Effects of Alcohol.**—Those who are best able

to judge assert that alcoholic mothers are more liable to various sorts of obstetric operations which necessarily increase the risk of the life of the fœtus. Moreover, such mothers are not favourable subjects for anæsthetics. "From the sterility among alcoholic women," says Professor Louise McIlroy, "and from the frequency of abortion and the difficulty of childbirth, one could see nature to a certain extent putting forward her efforts to reduce the number of children entering the world from the alcoholic stock."

This brings me to another point in connection with alcohol, viz., **the selective action of alcohol in the improvement of the racial stock**.

Pearl and Stockard have shown that parental alcoholism, by killing off the weaker ova in the various stages of development, eliminates the unfit specimens and thus ultimately benefits the race (see Tables XIII and XV). Such a contention, however, cannot be treated seriously from the point of view of human ante-natal hygiene, because, on the same line of argument, we ought to shut up our general hospitals, demolish our isolation hospitals, destroy our sanatoria and encourage slums, overcrowding, as well as everything that helps to increase general and infantile mortality.

TABLE XIX.

Nature of mating	Families	Pregnancies	Average number of pregnancies per family	Total births including stillbirths	Average number of births per family	Total miscarriages and abortions	Percentage of abortions &c., in total number of pregnancies
♂ × ♀	8,541	43,384	5·08	42,543	4·98	841	1·94
♂ × ♀	611	2,767	4·53	2,702	4·42	65	2·35
♂ × ♀	581	2,388	4·11	2,321	3·99	67	2·81
♂ × ♀	122	492	4·03	473	3·88	19	3·86
Total	9,855	49,031	4·98	48,039	4·87	992	2·02

♂ × ♀ = both parents free from tuberculosis.
♂ × ♀ = father tuberculous, mother free.
♂ × ♀ = mother tuberculous, father free.
♂ × ♀ = both parents tuberculous.

Tuberculosis.—Whilst cases of the transmission of the tubercle bacillus from mother to fœtus through the placenta have been recorded, they are so extremely rare as not to be worth con-

sidering from the practical point of view. But statistics recently collected by Kacprzak[1] show that there is a greater percentage of abortions amongst the tuberculous as compared with the non-tuberculous, as Table XIX shows :—

Other Toxic Agents that pass through the Placenta.—In addition to syphilis and alcohol, it has been proved that morphine, nicotine,[2] lead and other salts, and various therapeutic agents pass from the maternal into the fœtal circulation, and have therefore an important bearing on pre-natal hygiene and therapeutics. Also the toxins of infectious diseases may pass from the mother to the fœtus—even if the mother is herself protected by previous attacks or vaccination. Thus, according to Ballantyne, a pregnant woman who had small-pox may associate with cases of small-pox with impunity to herself, but with considerable risk to her unborn child. Conversely, vaccination of the mother may confer immunity upon the fœtus.

The abortifacient action of lead is well known. Indeed diachylon, which is a preparation of lead, is frequently being utilised for the purpose by the professional abortionist. Its passage through the placenta has been proved by means of experiments on animals, and Lewin[3] has published an interesting case of premature birth in a case of lead poisoning, in which nearly one-sixth of the weight of the fœtal liver was due to lead. Weller[4] studied the effect of lead on guinea-pigs. He found that when a normal female guinea-pig was mated with a male animal treated with lead, there was either complete sterility, or, when pregnancy resulted, the young were subnormal in weight and had a high percentage of early post-natal mortality. When a normal male was mated with a treated female, the result was sterility. Frongea (quoted by Whitridge Williams) finds that in Sardinia 20 per cent. of the married women working in lead are sterile, and another 23 per cent. have only one child.

[1] Martin Kacprzak, "Tuberculosis and Fertility," *Amer. Journ. Hygiene*, 1924, iv, p. 605.

[2] Nice, L. B. "Comparative Studies on the Effect of Alcohol, Nicotine, Tobacco Smoke and Caffeine on White Mice," *Journ. Exp. Zool.*, 1912, xii, pp. 133-152, and 1913, xiii, pp. 123-151.

[3] "Die Wirkung des Bleis auf die Gebärmutter," *Berl. klin. Wochenschr.*, 1904, xli, 1074-78.

[4] "The Blastophoric Effects of Chronic Lead Poisoning," *Journ. Med. Res.*, 1915, xxxiii, pp. 271-293.

The following Table, XX, showing the influence of lead absorption in the mother upon the fœtus, is given by Constantin Paul [1] :—

TABLE XX.

Observations	Pregnancies	Abortions	Premature births	Still-births	Dead infants and children	Living
(1) Four women ..	15	10	2	1	1	1
(2) Five women ..	36	26	1	2	5	2
(3) One woman ..	5	5	—	—	—	—
(4) Seven women ..	32	11	—	1	18	2
(5) Six women ..	29	8	1	—	12	8
Total ..	117	60	4	4	36	13

Notes.—The five women in Group 2, before working in lead, had five normal pregnancies.

The woman in Group 3, after ceasing work in a type foundry, had a healthy living child.

The women in Group 4 had their husbands suffering from plumbism.

The women in Group 5 presented only one sign of plumbism, i.e., the blue line on the teeth.

Of the 36 deaths during childhood, 20 died during first year, 8 in the second year, 7 in the third year, and 1 in the fourth year of life.

The **Report of British Factory Inspection Department,** 1897, shows that of:—

Seventy-seven married women who worked in lead, 15 never became pregnant, 15 others never bore a living child.

Of the 62 who became pregnant, there were 212 pregnancies, resulting in only 61 living and surviving children, 21 stillbirths, 90 miscarriages, 40 children born alive but dying immediately after.

According to Tardieu, out of 1,000 pregnancies in lead workers there were 608 premature births.

The effect of lead acetate administration to rabbits and fowls was studied by Cole and Bacchuter.[2] In rabbits they find the mortality of the young during the first four days after birth to be 47·7 per cent. in offspring of treated males as compared with 29·2 per cent. in those of normal males. Also the average birth weight was 48·9 grm. in the former, as compared with 59 grm. in the latter.

[1] Constantin Paul, *Arch. Gén. de Méd.*, 1860, i, pp. 513-533; also Thèse de Paris, 1861.

[2] *Proc. Soc. Exp. Biol. and Med.*, 1914.

By an order of the Privy Council, dated April 27, 1917, lead, in combination with other substances, whether sold as diachylon or under any other name—except machine-spread plasters—is declared to be a poison and can only be sold on the prescription of a doctor.

The Toxæmias of Pregnancy.—These conditions are generally believed to be brought about by toxins coming from the disintegration of the uterine tissue caused by ferments of the chorionic villi—ferments whose action is primarily intended for the purpose of facilitating the anchoring of the ovum at the placental site. These toxins, which are carried into the maternal circulation, are, under normal conditions, either destroyed by the liver, or sufficiently neutralised by antibodies manufactured inside the mother's blood, and then eliminated by the kidneys, bowels and other excretory organs. Hence, if the liver or excretory organs are defective, a state of pregnancy toxæmia results with the death of the fœtus by suffocation. Dental caries, which, by the way, is a frequent complication of pregnancy owing to the demand of the fœtus for calcium, and pyorrhœa, give rise to toxins which throw an extra burden on both the liver and kidneys, and hence great factors in the prevention of fœtal death due to true toxæmia of pregnancy are attention to oral hygiene, correction of constipation, and of renal deficiency by means of rest, diet, &c., and the treatment of the condition as soon as diagnosed.

Eclampsia and Fœtal Death-rate.—Out of 814 cases of eclampsia analysed by J. N. Cruickshank,[2] 235, i.e., 28·8 per cent., ended in the birth of a living mature child. In 87, i.e., 10·6 per cent., the child was alive but premature. In 252, i.e., 30·9 per cent., the child was stillborn (119 being mature and 133 premature but viable). Of the remaining cases, 20 resulted in miscarriage or abortion, 95 were dismissed from hospital undelivered, and 125 were delivered before admission into hospital. This last group, on account of the scantiness of information, has been omitted from the investigation.

Stillbirths were about three times as common in eclamptic cases as in unselected hospital cases. Premature birth was also distinctly more common in eclamptic cases.

Eclampsia is twice as common in primiparæ as in multiparæ.

The fœtal mortality has a definite direct relation to the number of fits. The greater the number of fits the higher the fœtal mortality.

[2] *Journ. Obst. and Gynæc. Brit. Emp.*, 1923, xxx, p. 541.

The causes of fœtal death in eclampsia are: –

(1) Placental infarction, throwing a large part of the placenta out of action.

(2) Direct action of the maternal toxin upon the fœtus, as is evidenced by the facts that cellular degeneration is sometimes found in the fœtal renal tubules and liver lobules. This cause of death is probably uncommon.

(3) Obstetrical measures adopted for the relief of the mother, such as prolapse of cord after the introduction of a Champetier de Ribes bag, or intracranial injuries resulting from hurried forceps delivery.

The following figures of fœtal deaths in eclampsia are given by Aleck W. Bourne[1]:—

TABLE XXI.

	No. of deaths	Mortality
Fœtal death	156	35 per cent.
Neo-natal death..	50	11 ,,
Survival	242	54 ,,
Total ..	448	100 per cent.

Umbilical Knots.—Browne found that whilst in an unknotted cord fluid will pass through the umbilical vein at a pressure of 10 mm. Hg, a cord with a *slack* knot required a pressure of 20 mm. to pass fluid through its vein. With two slack knots a superimposed pressure of 60 mm. Hg was required.

With a single knot and stretched by weights at the end the following pressures were needed:—

Weight 20 grm.	=	Pressure	50 mm.
,, 50 ,,	=	,,	100-110 mm.
,, 140 ,,	=	,,	140-150 ,,
,, 160 ,,	=	,,	165-170 ,,

The arterial pressure in the umbilical cord varies from 39·3 to 83·7 mm., while the venous pressure is about 16 mm.

It will be thus seen that umbilical knots may cause partial or total obstruction of the fœtal circulation.

Undetermined Causes of Ante-natal Deaths.—It is possible that some of these cases may be explained on the supposition

[1] "Recent Advances in Obstetrics and Gynæcology," London, 1926, p. 30.

that certain zygotes do not possess the power of developing beyond a certain stage of ante-natal life (Robinson).

Prevention of Ante-natal Deaths.—At present our power to prevent ante-natal death is practically limited to cases of albuminuria and syphilis.

Prevention of Intra-natal Deaths.—It is with respect to intra-natal deaths that the preventive methods are most successful.

Prevention of Asphyxia Neonatorum.—(1) Cephalic version in breech cases, especially in primiparæ. (2) Induction of labour about ten days before term in suitable cases. (3) Proper ante-natal supervision and intra-natal auscultation of the fœtal heart.

Prevention of Cerebral Hæmorrhage.—(1) Cephalic version in breech cases, especially in primiparæ. (2) Induction of labour in suitable cases. (3) No undue haste in delivery of after-coming head.

Effect of Birth-rate upon the Fœtal Death-rate.—Dr. Bolt rightly says that: "While the birth-control enthusiasts would indignantly disclaim any connection between the 'contraceptive methods' of 'voluntary parenthood' and induced abortion, it is very evident that the more 'moral technique' of contraception must often break down, and relief from the 'accidents' which follow be frequently sought in induced abortion." This is a statement which every medical practitioner of experience will emphatically endorse. Indeed in Armenia, where instruction in birth control is given through the maternity and infant welfare centres, provision is made by law for institutions where abortion may be carried out,[1] and I am informed that the same is the case in Soviet Russia.

Effect of Employment of Mother upon Stillbirth-rate.—The following statistics, given by Dr. R. M. Woodbury, and quoted by Josephine Baker, show that a mother's employment is associated with high stillbirth-rate :—

TABLE XXII.

Condition of mother with regard to employment	Stillbirth-rate (per cent.)
Employed away from home	4·9
Employed at home	3·5
Not employed	3·1

[1] *The International Year Book of Child Care and Protection*, London 1925, p. 341.

The effect of poverty upon stillbirth-rate is shown in the following table, given by Hersch[1]:—

TABLE XXIII.

Classes of arrondissements	Absolute figures		Stillbirths per 100 living births
	Stillbirths	Living births	
I (Richest)	1,004	12,313	8·2
II	1,390	19,998	7·0
III	7,279	82,821	8 8
IV (Poorest)	3,024	30,853	9·8
Paris	12,697	145,985	8·7

This table shows that poverty, *per se*, has no influence upon the stillbirth-rate—which is 8·2 per 100 live births in the richest class and 9·8 in the poorest.

Influence of Age of Mother upon the Stillbirth-rate.—According to Whipple[2] younger mothers have a higher stillbirth-rate than older ones. Thus:—

Age of mother.		Stillbirths per 100 live births
Under 20 years	..	11·1
20-24 ,,	..	4·0
25-29 ,,	..	5·1
30-39 ,,	..	4·4
Over 40 ,,	..	3·3

This means that a high stillbirth-rate is associated with first pregnancies (see also Table XLI, p. 139).

The stillbirth-rate is higher for illegitimate than legitimate children, especially among mothers less than 20 years old.

R. J. Ewart[3] collected statistics of 1,909 mothers, ranging in age between 16 and 49 years, amongst whom there were 726 fœtal deaths (including miscarriages and stillbirths) and found that the correlation coefficient between the age of the mother and the miscarriage rate in the family is $0{\cdot}2886 \pm 0{\cdot}018$, which is not only significant, it being sixteen times its probable error, but also of considerable size.

Influence of Ante-natal External Environment upon the Life and Welfare of the Fœtus.

A question of considerable sociological importance is the influence of maternal environment upon the welfare of the fœtus.

[1] Hersch, L. "L'inégalité devant la mort d'après les statistiques de la Ville de Paris, &c.," *Rev. d'Econ. Pol.*, 1920. Nos. 3 and 4 (quoted by Raymond Pearl, "The Biology of Death," Philadelphia and London, 1922, p. 205).

[2] George Chandler Whipple, "Vital Statistics," New York, 1923, p. 395.

[3] R. J. Ewart, "The Influence of the Age of the Parent on the Vitality of the Child." *Journal of Hygiene*, 1914, xlv, p. 453.

Are we justified in saying that such factors as poverty, underfeeding, mental strain, overcrowding, industrial occupation of the expectant mother, bad housing, &c., which have a very large influence upon the mother as well as some influence upon the infant after birth, exercise the same baneful influence upon the unborn fœtus? Let us consider these factors separately.

Poverty and Underfeeding.—The philanthropist anxious to do good will quite naturally contend that poverty and malnutrition of the mother are bound to affect the fœtus adversely. On the other hand, a consideration of the facts with regard to the manner in which the fœtus derives its nourishment would lead one to expect no relationship between these conditions of the mother and the welfare of her unborn child. The most reliable experimental evidence from animals, as well as statistical data in man, confirms one's theoretical expectations.

Animals experimentally starved produce well-nourished fœtuses, and exact statistics collected by Lotte Landé,[1] in Germany, and by Miss Bruce Murray,[2] in this country, show that infants born under different economic conditions have practically the same weights and lengths, as the following tables show (see Tables XXIV-XXVIII).

TABLE XXIV.—THE RELATIONSHIP BETWEEN SOCIAL CONDITION OF MOTHER AND BIRTH WEIGHT AND LENGTH (Miss Bruce Murray).

Mother's income and baby's		(a) Length.	(b) Weight
1914. Pre-war	r =	0·017 ± 0·051	0·012 ± 0·051
1915.	r =	0·073 ± 0·050	0·095 ± 0·050
1918.	r =	0 029 ± 0·042	0·031 ± 0·040

TABLE XXV.—CORRELATION BETWEEN MATERNAL HEIGHT AND BABY'S.

	Length	Weight
Boys ..	r = 0·203 ± 0·039	r = 0·123 ± C·049
Girls ..	r = 0·121 ± 0·046	r = 0'110 ± 0·046

[1] Entwicklung und Schicksal der im Kaiserin-Auguste-Victoria Haus-geborenen Kinder. *Zeitschr. f. kinderheilk.*, 1919.

[2] "The Effect of Maternal Social Conditions and Nutrition upon Birth Weight and Birth Length." Med. Res. Council, Spec. Rep. Ser. No. 81.

It is only in case of correlation between height of mother and length of her infant son that $\sigma > 3$ P.E. and is therefore significant.

Correlation between **maternal age and baby's weight** is *nil* (= 0·004 ± 0·017).

TABLE XXVI.—AVERAGE BIRTH WEIGHT AND LENGTH FOR ALL CLASSES.

Year	Average weight (lb.)	Average length (in.)
1914	7·16	19·93
1915	7·16	20·07
1918	7·11	20·20

TABLE XXVII.—INFLUENCE OF MATERNAL ENVIRONMENT UPON THE FŒTUS AS SHOWN BY BIRTH WEIGHT AND BIRTH LENGTH.

(1) *German Statistics on Birth Weight* (*Lotte Landé*).

Birth weight	Before war (1909-1913)	During war (1915-1918)
Under 2,500 grm. ..	42 = 4·8 per cent.	27 = 3·4 per cent.
2,500-2,990 ,, ..	133 = 15·1 ,,	125 = 15·5 ,,
3,000-3,490 ,, ..	383 = 43·5 ,,	345 = 42·8 ,,
3,500-3,990 ,, ..	274 = 31·1 ,,	242 = 30·0 ,,
4,000 grm. and over ..	49 = 5·6 ,,	68 = 8·4 ,,
Total ..	881 = 100 per cent.	807 = 100 per cent.

TABLE XXVIII.

(2) *English Statistics on Birth Weight and Length* (*Miss Bruce Murray*).

Year	Average weight in lb.				Average length in in.			
	A	B	C	D	A	B	C	D
1914	7·25 ±0·069 (101)	7·15 ±0 071 (100)	7 10 ±0·119 (37)	7·10 ±0·061 (106)	20·06 ±0·052 (101)	19·92 +0·052 (101)	19·85 ±0·092 (37)	19·94 ±0·054 (105)
1915	7·29 ±0 053 (123)	7·05 ±0·059 (114)	7·23 ±0·125 (26)	7·37 ±0·058 (104)	20·24 ±0·049 (123)	20 05 ±0·049 (113)	20·26 ±0·108 (26)	20·28 ±0·058 (104)
1918	7·10 ±0·052 (138)	7·15 ±0·046 (226)	6·95 ±0·178 (18)	7·01 ±0·047 (140)	20·27 ±0·016 (137)	20·16 ±0·035 (225)	20·22 ±0·145 (18)	20·30 ±0·045 (140)

Classes A, B, C are in descending economic order (all being of hospital class).

Class D.—Unmarried mothers "having not only much mental distress and anxiety as the result of their position, but a really hard struggle to make ends meet and provide for themselves during the last three months of pregnancy."

The numbers in brackets represent the numbers of cases investigated.

It cannot even be said that a mother poorly nourished during pregnancy will be unable to nurse her newly-born baby, because statistics show that the neo-natal mortality is practically identical amongst all classes of society.

Mental Stress.—Miss Murray's statistics of legitimate and illegitimate children, whose mothers had much mental distress and anxiety as the result of their position, show no appreciable difference in the birth weights and lengths of the two classes of babies.

Housing.—There are no direct statistics upon the subject of housing and fœtal welfare, but on theoretical grounds we would expect bad housing conditions with improper ventilation to have very little deleterious effect upon the fœtus, *unless the ventilation is really so bad as to cause partial asphyxia of the mother*, for it is only in states of asphyxia that the mother has been found in experimental animals to take some of the oxygen from the fœtus.

The Influence of Ante-natal External Environment upon the Life and Welfare of the Child during its Early Post-natal Life is shown in the following table:—

TABLE XXIX.—INFANT MORTALITY IN BOROUGHS WITH LOWEST AND HIGHEST POOR RATES.

Age	Hampstead	Lewisham	Woolwich	Bermondsey	Finsbury	Shoreditch
Under 1 week	21·1	17·0	20·0	19·8	21·2	21·6
2nd week	4·5	5·1	4·2	3·6	5·5	6·5
3rd week	3·0	4·0	3·8	4·9	8·2	6·8
4th week	1·5	1·4	3·8	2·5	4·3	2·3
Under 1 month	30·1	27·5	31·8	30 8	39·2	37·2
0-3 months	44·4	44·3	48·0	55·4	65·2	68·8
4-6 ,, ...	13·6	13·3	10·0	29·0	29·8	29·9
7-9 ,, ...	6·8	7·1	12·8	21·3	21·6	28·2
10-12 ,, ...	3 8	10·8	8·3	23·9	20·0	23·9
0-12 ,, ...	68·6	76·5	79·1	129·6	136·6	156·8

During the first week the mortality rates are practically identical in Hampstead and Shoreditch.

During the first month mortality in Shoreditch is only about 25 per cent. higher than that in Hampstead.

But during the age period seven to nine months the mortality in Shoreditch is 400 per cent. higher than that in Hampstead, and during the age period ten to twelve months 600 per cent. higher.

If external maternal environment had any influence upon the fœtus we ought to find the influence manifesting itself in the early weeks of post-natal life and to decrease as the child gets older. Hence we see that ordinary pre-natal environment is not a factor in the causation of excessive neo-natal mortality.

The table from Hersch (already quoted on p. 122) giving the various stillbirth-rates in different arrondissements of Paris, arranged in order of economic conditions, illustrates the same thing.

Clinical observation in the case of **maternal heart disease** shows that unless the heart action is sufficiently bad to cause asphyxia of the mother, the pregnancy continues to full term with the birth of a living child.

The industrial occupation of the mother has been stated to exercise a baneful influence upon the fœtus; for apart from the fact that certain occupations are dangerous during the expectant period, e.g., lead, phosphorus, tobacco, &c., statistics have been presented to show that the mortality of infants whose mothers worked in factories right up to the time of labour was nearly 500 per 1,000 during the first week of life, compared with 85 per 1,000 in infants of the same age whose mothers lived in the country and worked on farms. When the mothers of the first class stopped work a month before confinement, their physical condition improved and they were able to suckle their babies. I believe that these findings want thorough confirmation.

Maternal Impressions.—The idea that certain impressions occurring to the mother during pregnancy have an influence upon the future child has no scientific foundation (see Chapter IX).

Influence of Heredity upon Fœtal Welfare.—Even after we have eliminated all the various factors which produce preventable fœtal deaths, there will still remain a certain percentage of fœtuses who will perish either before birth or during the neo-natal period from causes which are associated with heredity, or some factor of unknown origin. Indeed, statistics collected in *the same town* (Boston) of the neo-natal mortality among infants of native-born and foreign-born mothers show that congenital debility and malformations, which are probably of hereditary origin, vary in incidence with the nationality or race of the mother as follows:—

Table XXX.

Nationality of mother	Neo-natal mortality from congenital debility and malformation
Native American	50 per 1,000 births
Canadian	31 ,, ,, ,,
Irish	49 ,, ,, ,,
Italian	24 ,, ,, ,,
Russian (mainly Jewish)	20 ,, ,, ,,

Contracted Pelvis as a Cause of Fœtal Death.

The following figures, expressing the experience of Queen Charlotte's Hospital during the three years 1920-1922, are given by Bourne :—

Table XXXI.

Method of delivery	No. of cases	Fœtal deaths	Mortality
(1) Forceps without induction ..	38	11	29·0 per cent.
(2) Craniotomy	18	18	100·0 ,, ,,
(3) Induction	226	34	15·0 ,, ,,
(4) Cæsarean section	117	11	8 6 ,, ,,
Total ..	4.9	74	18·5 per cent.

If we compare Groups 1 and 2, in which no ante-natal supervision took place, with those of 3 and 4, in which there was proper supervision, we obtain the following figures :—

Method of treatment of contracted pelvis	No. of cases	Fœtal deaths	Mortality
No ante-natal care	56	29	51·8 per cent.
With ante-natal care	353	45	12 8 ,, ,,

The effect of obstetrical procedure on the fœtus is seen from the following Table XXXII (Bourne) :—

Table XXXII.

	Total	Survived	Died	Mortality
Natural or unassisted delivery	88	62	26	29 6 %
Induction	41	18	23	56·1 ,,
Cæsarean section	74	57	17	23·0 ,,
Accouchement forcé	3	—	3	100·0 ,,

Obstructed labour, such as occurs in cases of contracted pelvis or similar cause, not only causes a high intra-natal mortality, but is also responsible for a considerable portion of early neo-natal deaths (those occurring within a few days of birth). Moreover, a percentage of the infants that survive remain permanently crippled, either physically or mentally or both, as the result of intracranial hæmorrhage during birth. Hence, ante-natal care would not only save a large number of potential citizens from death every year, but would also be a material help in reducing the number of infantile hemiplegics and mentally-defective children.

Dr. F. S. Kellog, of the Boston Lying-in Hospital, gives the following figures showing the **incidence of the various complications of pregnancy** :—

Total number of cases	4,996	
Total abnormal cases	1,524	= 30 per cent.
Albuminuria alone	361	= 7 ,,
,, + raised blood-pressure	259	= 5 ,,
,, ,, ,, + symptoms of toxæmia	195	= 4 ,,
Contracted pelvis	401	= 8 ,,
Heart disease	111	= 2 ,,
Pulmonary tuberculosis	10	= 0·2 ,,
Ante-partum hæmorrhage	33	= 0·7 ,,
Pyelitis	20	= 0·4 ,,
Syphilis	21	= 0·4 ,,
Gonorrhœa	10	= 0·2 ,,
Chronic nephritis	5	= 0·1 ,,
Diabetes	3	= 0·06 ,,
Ovarian cysts, fibroids, &c.	30	= 0·6 ,,

We see therefore that 30 per cent. of all cases of pregnancy present some sort of abnormality which may in the large majority of the cases be prevented. The only way to ensure ante-natal care to these 30 per cent. of cases is to give it to all expectant mothers.

The Value of Ante-natal Supervision.—The statistics given by Ballantyne, as well as the American statistics given in Table XXXIII, show that energetic ante-natal care has been instrumental in reducing the stillbirth-rate by about 21 per 1,000 live births, and that of neo-natal deaths by 18 per 1,000 live births, giving a total number of lives that could be saved in 1923 in England and Wales of $(18 + 21) \times 760 = 30{,}000$. The statistics of Dr. Meyer of the annual death-rates from "congenital causes" during the decennium, 1910-1919, for New York City, as a whole the

Borough of Manhattan and the Bronx, and New York City outside this borough, show that in Manhattan and the Bronx, where intensive ante-natal supervision is being carried out, the death-rate from congenital causes fell by 17·6 per 1,000, although in New York City outside that borough the mortality rate from that cause increased by 12·7 per 1,000, and in New York City as a whole it remained practically stationary.

Dr. Dublin's statistics, comparing New York City as a whole in 1920, with 8,743 expectant mothers supervised by the Maternity Centre Association during a period of twenty-seven months ending August 15, 1921, show the following facts, viz., a reduction in the stillbirths, prematurity and congenital malformation rates as the result of efficient ante-natal supervision.

TABLE XXXIII.—RESULTS OF ANTE-NATAL SUPERVISION IN NEW YORK.

Causes of death	New York City, 1920	Maternity Centre Association, New York City, 1919-1921
Stillbirths	46·5 per 1,000 births	25·1 per 1,000 births
Prematurity	14·7 ,, ,, ,,	4·8 ,, ,, ,,
Congenital malformations ..	3·5 ,, ,, ,,	1·6 ,, ,, ,,
Maternal mortality	6·07	1·33

TABLE XXXIV.—RESULTS OF ANTE-NATAL SUPERVISION IN TORONTO. (Fitzgerald.)

	Semi-private cases (not supervised)	Public ward cases (not supervised)	Public ward cases (supervised)
Number of cases ..	1,198	505	461
Deaths of mothers ..	10 (0·8 per cent.)	18 (3·5 per cent.)	2 (0·4 per cent.)
Stillbirths	45 (4·0 ,, ,,)	40 (7·9 ,, ,,)	6 (1·3 ,, ,,)
Eclampsia	20 (1·6 ,, ,,)	16 (3·0 ,, ,,)	2 (0·4 ,, ,,)

English Statistics.

H. Harvey Evers[1] gives an account of the results of ante-natal supervision in Newcastle-upon-Tyne. The following is a tabular statement of the results:—

[1] *Public Health*, November, 1926.

TABLE XXXIVa.—RESULTS OF ANTE-NATAL SUPERVISION IN NEWCASTLE.

	Number of cases	Treated		Untreated	
		Number of cases	Still-births	Number of cases	Still-births
1. Malpresentations ..	57				
(*a*) Breech	14	11	0	3	2
(*b*) Occipito-posterior ..	40	28	0	12	1
(*c*) Oblique	3	3	0	—	—
2. Ante-partum hæmorrhage	13				
(*a*) Placenta prævia ..	4	4	3	—	—
(*b*) Accidental hæmorrhage	9	9	3	—	—
3. Albuminuria	15				
(*a*) Symptomless ..	4	4	0	—	—
(*b*) Œdema	4	4	0	—	—
(*c*) Marked symptoms ..	7	7	1	—	—
4. Contracted pelvis ..	20	20	0	—	—
5. Venereal disease ..	4	4	0	—	—
Total	109	94	0	15	3

TABLE XXXV.

Statistics of Stillbirths, showing Value of Ante-natal Care.

EDINBURGH STATISTICS (Ballantyne).

For the whole city (432 out of 9,028 births) ..	47·8 per 1,000 births
For practice of Royal Maternity Hospital (156 out of 2,770 births)	56·3 ,, ,, ,,
(i) Internal department (104)	68·8 ,, ,, ,,
(ii) External department (52)	41·2 ,, ,, ,,
(iii) V.D. department	157·8 ,, ,, ,,
(*a*) Receiving ante-natal treatment (7 in 138)	50·7 ,, ,, ,,
(*β*) Not receiving ante-natal treatment (20 in 33)	606·0 ,, ,, ,,
(iv) Non-V.D.[1] cases receiving ante-natal treatment (40 in 678)	5·9 ,, ,, ,,
(v) V.D. + Non-V.D. cases receiving ante-natal supervision and treatment (110 in 816)	13·5 ,, ,, ,,

Note the extremes: V.D. not supervised .. 606 per 1,000
Non-V.D. supervised .. 5·9 ,, ,,

[1] "V.D." stands for venereal disease.

TABLE XXXVI.—THE INCIDENCE AND PREVENTABILITY OF THE VARIOUS CAUSES OF FŒTAL MORTALITY (Eardley Holland).

Cause of fœtal death	Incidence per cent. of total mortality	Preventability			
		Ante-natally per cent. of total mortality	Intra-natally per cent. of total mortality	Ante- and Intra-natally per cent. of total mortality	Total per cent. of total mortality
Complications of labour	51	1	20	9	30
Syphilis	16	16	—	—	16
Toxæmia of pregnancy ..	10	3	—	3	6
Unknown	10	—	—	—	—
Placental insufficiency ..	6	—	—	—	—
Fœtal deformity.. ..	5	—	—	—	—
Maternal	2	1	—	—	1
Totals ..	100	21	20	12	53

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CHAPTER V

CHILD MORTALITY

"The difference between the death of children and that of old people is the same as that between the plucking of unripe and of ripe figs; the former is bad for both the fruit and the tree, the latter is good for both."—THE MIDRASH.

CHILD mortality may be conveniently divided into: (1) Mortality during the first year of life; (2) mortality during the subsequent four years.

Infant Mortality.—The term infant has not the same meaning in vital statistics as it has in common law or in ordinary parlance. Any person under the age of 21 is in English law considered an infant; and from the point of view of the provisions of the Children Act, 1908, an "infant" is a child under 7 years of age; whilst in common parlance the term infant has, of course, a very elastic meaning. In vital statistics, however, it is generally agreed that **an infant is any child from the moment of birth up to the end of the first year of life.** By **infant mortality,** therefore, we understand **the death-rate of children under the age of 1 year.**

The Measurement of Infant Mortality.—From what we have said before (see p. 104) it will be realised that the most logical method of measuring infant mortality rate would be by means of the specific death-rate for the age-group 0-1 year, i.e., by the annual number of deaths of infants under 1 year old per 1,000 infants of the same age during the same period. But as a matter of fact the specific death-rate for infants is hardly ever used for this purpose, because it is very difficult to ascertain, with any degree of accuracy, the average number of infants alive during any calendar year (i.e., the number alive at the middle of the year). The only ways of doing it approximately are either by taking a census of the infant population on the 1st of every July, or by keeping a monthly record of births and deaths of infants under 1 year. But even such laborious undertakings

would not give satisfactory results, because infants' ages are often recorded incorrectly. For these reasons the **infant mortality rate** is practically universally measured by the **annual number of deaths of infants under 1 year old per 1,000 infants born alive during the same year**, i.e. :—

Infant mortality rate =

$$\frac{\text{Number of infants under 1 year dying in one year}}{\text{Number of infants born alive during the same year in the same area}} \times 1{,}000$$

It is to be noted that stillbirths are to be excluded not only from the denominator but also from the numerator. Moreover, the ratio does not tell us how many infants born alive during any one year fail to reach their first birthday, for the numerator and the denominator do not deal with the same infants. Further still, this method of measuring infant mortality, though the most convenient, is not free from errors, because the age of a baby at death is not always correctly given, as infants just under a year old are often stated as having reached that age.

Real and Apparent Infant Mortality Rates.—In the comparative study of infant mortality rates in different parts of the world, or even in the same geographical area, at different times, great caution is necessary not to mistake an apparent for a real infant mortality rate. For since the denominator of the ratio is the number of live births during the year under consideration, it is obvious that there may be an apparent difference in the infant mortality rates in two successive years in the same locality, entirely due to an annual fluctuation in birth-rates, so that during the second year after mobilisation for war, when the birth-rate is low, the infant mortality may appear higher than it actually is, whilst in the years immediately succeeding war the infant mortality rate may appear to be much lower than it really is on account of the increase in birth-rate. Similarly, in areas where birth registration is not perfect, the infant mortality may appear higher than it actually is, and with improvement in birth registration there may be an apparent reduction in infant mortality, although in reality not a single infant's life has been saved. In England and Wales birth registration has been perfect for a large number of years, and therefore, but for the natural fluctuation in birth-rate, we can say that every difference in infant mortality rates indicates a real saving of infants' lives, but in those parts of the world where birth registration is not complete we must not hastily conclude that a reduction in the death-

rate of infants is due to any successful campaign to save infants' lives until we are sure that the reduction is not entirely to be accounted for by an improvement in birth registration. Thus, supposing that in a certain year out of a thousand infants born alive only 800 were registered, and during the same year eighty infants died, the apparent infant mortality is $\frac{80}{800} \times 1{,}000 = 100$. At a subsequent date all the 1,000 infants born alive were registered and eighty infants died; the infant mortality is now $\frac{80}{1{,}000} \times 1{,}000 = 80$ only, so that although not a single infant life has been saved the infant mortality rate has apparently decreased from 100 to 80, i.e., by 20 per cent.

The following is a concrete example.[1] In Baltimore the following were the birth and infant mortality rates for 1910 and 1915:—

Year	Registered birth-rate (per 1,000 population)	Infant mortality (per 1,000 registered births)
1910	17·6	218
1915	23·3	120

The apparent fall in infant death-rate between 1910 and 1915 is

$$\frac{(218-120)}{218}\ 100 = 45 \text{ per cent. of that in 1910.}$$

But this is quite certainly not the true magnitude of the fall, since, while a census has shown that the total increase in the population during the interval was only 4 per cent., the apparent rise in the birth-rate during the same time was $\frac{(23{\cdot}3-17{\cdot}6)}{17{\cdot}6}\ 100 = 32$ per cent. of that in 1910.

It is, therefore, certain that part of the decline in the infant mortality rate was apparent only and due to an improvement in birth registration.

Assuming that the birth-rate in 1910 was really as high as in 1915, viz., 23·3, then the true infant mortality rate in that year was $218 \times \frac{17{\cdot}6}{23{\cdot}3} = 165$ approximately, and therefore the real decline in the death-rate of infants was only $\frac{(165-120)}{165}\ 100 = 27$ per cent. and not 45 per cent., as was apparently the case.

The Trend of Infant Mortality.—Although as early as 1538 Henry VIII ordered all parishes to keep registers of christenings, weddings and deaths, it is not till the seventeenth century that it

[1] See L. J. Dublin, "The Present Status of Birth Registration," *Quart. Journ. Amer. Statist. Assoc.*, March, 1917 (quoted by Newsholme).

occurred to anybody to analyse these records. It was in 1665 that Captain John Graunt, of London, published his "Natural and Political Observations . . . made upon the Bills of Mortality." He there points out the relatively high rate of mortality in the earliest years of life. In 1812 a law was passed in England making the registration of deaths compulsory, but it was not till later in the century that exact records of infant mortality rates became available. But although we cannot tell exactly what was the magnitude of the infant mortality rate during the seventeenth and eighteenth centuries, we can form some good idea of its size from certain historical facts. According to Justin McCarthy, Queen Anne had borne eighteen or nineteen children, all of whom, with the exception of the Duke of Gloucester who lived till the age of 11, died in infancy. Gibbon, the historian (born 1737), says in his "Memoirs" that he was the sole survivor of six sons, the other five having been "snatched away in their infancy." Percival, quoted by Holt, writing in 1789, says that "in Manchester half the children born die before reaching the fifth year," whilst in France, at about the same time, 50 per cent. of the children died before the end of the second year. According to Farr the following were the mortalities of infancy and early childhood during the eighteenth and early part of the nineteenth centuries :—

Period	1730-49	1750-69	1770-89	1790-1809	1810-29
Percentage deaths under 5 years	74·5	63·0	51·5	41·5	31·8

In the early part of the nineteenth century, after the introduction of vaccination, a considerable fall in infant mortality due to small-pox manifested itself. In 1860-61, according to Farr,[1] the infant mortality rates (per 1,000 births) were as follows: Sweden 141, Scotland 149, England 170, France 223. The following tables give the trend of infant mortality in England and Wales from 1871 to the present day, for France from 1892 to 1915, and for Sweden from 1751 onwards.

[1] William Farr, "Mortality of Children in the Principal States of Europe." *Journ. Statist. Soc.*, xxix, London, 1866.

TABLE XXXVII.—GENERAL MORTALITY AND AGE DISTRIBUTION OF INFANT MORTALITY IN ENGLAND AND WALES, 1871-1923.

Year	Infant mortality per 1,000 births						General death-rate per 1,000 persons
	Under 4 weeks	1-3 months	Total under 3 months	3-6 months	6-12 months	Total under 1 year	
1871-1880	—	—	—	—	—	149	21·4
1881-1885	—	—	67	28	44	139 } 142	19·1
1886-1890	—	—	69	30	46	145	
1891-1895	—	—	74	31	46	151 } 153	18·2
1896-1900	—	—	74	34	48	156	
1901-1905	—	—	70	28	40	138 } 128	15·4
1906-1910	40	23	63	22	32	117	
1911-1915	39	20	59	20	31	110	
1916	37	17	54	15	22	91	
1917	37	17	54	16	26	96	
1918	36	17	53	16	28	97 } 100	14·3
1919	40	15	55	13	21	89	
1920	35	16	51	12	17	80	
1921	35	15	50	14	19	83	12·1
1922	34	13	47	11	19	77	12·8
1923	32	11	43	10	16	69	11·6
1924	33	12	45	11	19	75	12·2
1925	32	13	45	11	19	75	12·2

The infant mortality rates for France between 1892 and 1915 are given below:—

1892-1895	258 per 1,000
1896-1900	229 ,,
1901-1905	164 ,,
1906	145 ,,
1909	143 ,,
1910	146 ,,
1915	145 ,,

The figures for Sweden show that till 1800 the infant mortality rates fluctuated between 200 and 222, but that from that time onwards the rate began to decline, until by 1900 it was only about 100, and in 1917 it dropped to 55. It is possible that part of the drop may be apparent only, and is to be accounted for by an improved birth registration. The most important point about infant mortality is that it is higher than the mortality during any other period of life.

TABLE XXXVIII.—INFANT AND CHILD MORTALITY IN SWEDEN, 1751-1900.

Period	Total death-rate per 1,000	Age-group in years			
		0-1 per 1,000 births	1-3 specific death-rate	3-5 specific death-rate	0-5 specific death-rate
1751-55	26·52	205·75	52·17	27·31	86·07
1756-60	28·25	203·41	40·50	26·26	81·64
1761-65	29·08	221·73	53·94	28·49	90·46
1766-70	26·38	210·41	50·12	27·06	85·14
1771-75	33·07	212·89	66·65	36·15	92·88
1776-80	24·86	192·02	56·21	29·13	83·74
1781-85	27·80	193·98	62·64	36·16	86·44
1786-90	27·61	205·70	48·02	23·47	79·55
1791-95	25·21	192·59	44·63	20·76	77·09
1796-1800	25·65	199·53	48·02	23·47	79 55
1801-05	24·35	186·08	41·48	18·70	70·65
1806-10	31·45	211·46	59·09	29·09	87·42
1811-15	27·11	191·76	56·46	20·57	81·54
1816-20	24·63	175·51	45·93	17·96	71·00
1821-25	22·07	158·85	36·24	14·33	61·63
1826-30	25·10	175·76	37·72	17·07	64·53
1831-35	23·05	167·31	33·44	14·44	60·32
1836-40	22·53	166·35	35·47	15·42	60·31
1841-45	20·20	153·77	30·38	14·39	56 18
1846-50	20·95	152·56	33·39	16·48	57·34
1851-55	21·65	148·89	35·32	18·79	58·83
1856-60	21·73	143·47	39·12	23·87	61·96
1861-65	19 76	136·17	40·95	21·78	58·48
1866-70	20·54	141·93	38·78	19·59	56·14
1871-75	18·28	133·57	29·78	14·64	51·47
1876-80	18·26	126·28	36·26	19·80	53·01
1881-85	17·53	116·08	31·82	17·09	47·18
1886-90	16·37	105·00	25·88	13·41	40·06
1891-95	16·61	102·76	23·97	12·65	38·21
1896-1900	16·12	100·50	21·78	9·72	36·65
1917	—	55	—	—	—

Causes of Infant Mortality.—The number of factors which are responsible for infantile deaths are so numerous and interwoven, that it is extremely difficult to pick out which is the important factor in any particular case. We may, however, broadly divide these causes into two main groups, viz. :—

(*a*) Uncontrollable.

(*b*) Controllable.

Under the **uncontrollable** group come such factors as geographical position, racial composition of the community, sex ratio at birth, the age distribution of the mothers, the percentage of illegitimate births, the percentage of first births, seasonal and possibly economical conditions. This is well seen from the tables presented herewith (XXXIX-XLI and XLVII-XLVIII), in which

it is shown clearly that marked variations occur in the infant mortality rates as the results of the operation of each of these uncontrollable factors. It is to be remarked, however, that with the exception of circumstances relating to geographical position and race, the death-rates depending upon the other factors are probably amenable to preventive treatment, and are therefore really controllable.

TABLE XXXIX.

Infant Mortality per 1,000 live births in Different Parts of the World, according to Sex.

Country	Mortality			Country	Mortality		
	M.	F.	P.		M.	F.	P.
Australia (1919) ..	76	62	69	Italy (1916)	175	158	166
Belgium (1912) ..	132	107	—	Jamaica (1919) ..	168	155	162
Ceylon (1919)	228	217	223	Japan (1916)	179	161	170
Chile (1918)	261	248	255	Netherlands (1917) ..	64	50	57
Denmark (1918) ..	82	66	74	New Zealand (1918) ..	54	43	48
England and Wales (1923)	78	60	69	Norway (1917) ..	71	57	—
				Russia (1909) ..	265	237	—
Finland (1918).. ..	123	108	—	Scotland (1919) ..	113	90	—
France (1919)	—	—	119	Spain (1915)	166	145	156
Germany (1918) ..	167	140	154	Sweden (1915) ..	84	67	76
India (1921)	—	—	197	Switzerland (1918) ..	97	79	88
Ireland (1918).. ..	96	77	86	United States (1919)..	96	79	87

M = male ; F = female ; P = persons.

In the same country the mortality varies greatly from town to town, partly as the result of differences in racial composition and partly as the result of uncontrollable differences, such as climate, soil, &c.

In the Uganda Protectorate, according to Dr. and Mrs. Albert R. Cook,[1] who have been working in that country for more than twenty years, the stillbirth-rate is 150 per 1,000 ; the incidence of syphilis amongst the adult female population is 66 per cent. ; the ante-natal mortality is 67 per cent. amongst expectant mothers suffering from syphilis ; out of 100 conceptions only 45 survive to the end of twelve months ; the infant mortality is 330 per 1,000 births, and they have found that skilled trained treatment in hospital reduces the mortality by 75 per cent.

When we come to the **controllable** group we approach a field which is being hotly contested by combatants of different shades of thought. There are some who attribute high infant mortality rates to lack of ante-natal care, maternal ignorance and lack of proper care in the rearing of infants, &c., and ascribe the whole of the great drop in recent years to the activities of the ante-natal and infant welfare centres, aided by such measures as the Midwives Act and the Notification of Births Act. Others—basing

[1] See *Maternity and Child Welfare*, 1923, vii, p. 261.

TABLE XL.

Influence of Race on Infant Mortality under same Climatic and certain other Environments in New York, 1915.

Nativity of mother	Total births	Total infant deaths	Infant mortality per 1,000 births	Deaths from congenital diseases
United States	36,992	3,935	106	54·4
Italy	29,717	3,068	103	29·5
Russia-Poland } mainly Jews	24,432	1,903	78	32·0
Austria-Hungary } mainly Jews	11.797	942	80	28·4
Ireland	5,027	600	119	53·0
Germany	1,903	220	116	57·7
England	486	67	138	47·4
Sweden	550	36	65	38·9
Bohemia	237	32	135	51·5
Scotland	202	16	79	—
Switzerland	45	10	222	65·2
France	100	8	80	59·1
Other and mixed (native and foreign)	29,768	5,029	102	—
Total white	—	—	99	—
Total coloured	—	—	181	—
Total all races	141,256	13,866	98	92·7

NOTE.—The exclusion of Jewish infants reduces the total births to 105,027 and the total infant deaths to 11,021. Hence the total mortality, exclusive of Jews, is raised from 98 to $\frac{11,021}{105,027} \times 1,000$, i.e., to 105.

TABLE XLI.

Age and Status of Mother as a Factor of Infant Mortality.

Age of mother	Total births	Live births	Infant deaths	Infant mortality rate	Stillbirths	
					Number	Per cent. of total births
All mothers	2,662	2,587	337	130·3	75	2·8
Under 20	112	108	28	259·3	4	3·6
20-24	737	725	93	128·3	12	1·6
25-29	853	833	95	114·0	20	2·3
30-39	840	809	105	129·8	31	3·7
40 and over	120	112	16	142·9	8	6·7

The relative number of first-born children has a distinct bearing, not only upon the number of infant deaths but upon the maternal mortality as well. First-born children have on the average a 33 per cent. higher mortality than the second-born.

themselves on the fact that in places where such activities did not take place till very recently, the decline in infant mortality was both coeval and commensurate with that in places where the campaign work has been in existence since the beginning of the century,—allege that the various Acts and the infant welfare activities have accomplished nothing, and that the fall in infant mortality is due entirely to general causes which are altogether unconnected with the intensive infant welfare campaign work. We shall see that the truth, as always, lies between these two extreme views.

TABLE XLII.—BIRTH-RATES, DEATH-RATES, AND INFANT MORTALITY RATES IN ENGLAND AND WALES, 1841-1924.

Period	Birth-rate	Death-rate	Infant mortality per 1,000 live births		Acts of Parliament
			Rate	Increase or decrease per cent.	
1841-1850 ..	32·6	21·6	153	—	—
1851-1860 ..	34·1	21·2	154	+ 0·6	—
1861-1870 ..	35·2	21·3	154	0·0	Education Act, 1870
1871-1880 ..	35·4	20·3	149	— 3·2	Public Health Act, 1875
1881-1885 ..	33·5	19·7	139	— 6·7	—
1886-1890...	31·4	18·5	145	+ 4·1	—
1891-1895 ..	30·5	18·5	151	+ 3·2	Housing of the Working-Classes Act, 1890
1896-1900 ..	29·3	17·5	156	+ 3·2	—
1901-1905 ..	28·2	16·0	138	— 11·5	Midwives Act, 1902
1906-1910 ..	25·3	14·4	117	— 15·2	Education Act, 1907. Notification of Births Act, 1907. Children Act, 1908
1911-1915 ..	23·6	13·8	110	— 5·9	National Insurance Act and Tuberculosis Order, 1911. Notification of Births (Extension) Act, 1915
1916-1920 ..	20·1	13·7	90	— 18·1	V.D. Regulations, 1916. Maternity and Child Welfare Act, 1918
1921 ..	22·4	12·1	83 } 76	— 15·6	—
1922 ..	20·4	12·8	77		
1923 ..	19·7	11·6	69		
1924 ..	18·8	12·2	75		
1925 ..	18·3	12·2	75		

The birth-rate in 1925 was the lowest recorded, except during the war years 1917 and 1918.

Analysis of Infant Mortality Statistics.—In order to estimate the relative importance of each of the factors which are believed

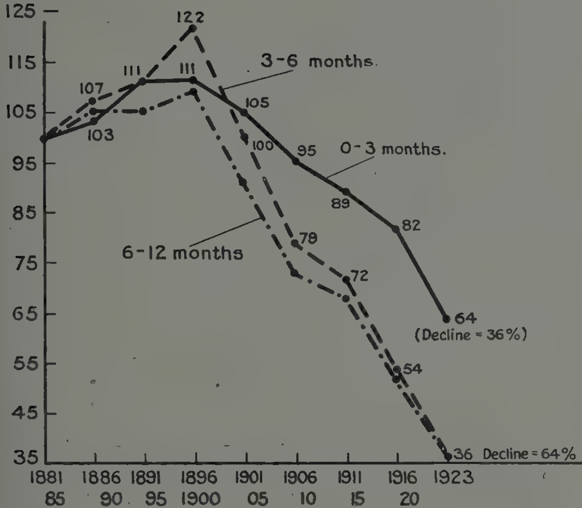
3-6 months.
0-3 months.
6-12 months
(Decline = 36%)
Decline = 64%
125
115
105
95
85
75
65
55
45
35
1881 85
1886 90
1891 95
1896 1900
1901 05
1906 10
1911 15
1916 20
1923
122
111
111
107
103
105
100
95
89
82
78
72
64
54
36

FIG. 30.

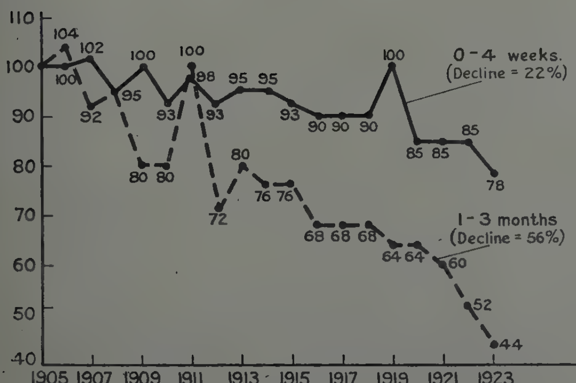
0-4 weeks.
(Decline = 22%)
1-3 months
(Decline = 56%)
110
100
90
80
70
60
50
40
1905 1907 1909 1911 1913 1915 1917 1919 1921 1923
104
102
100
100
100
100
98
95
95
95
93
93
93
92
90 90 90
85 85
85
78
80 80
80
76 76
72
68 68 68
64 64
60
52
44

FIG. 31.

to have an influence on the mortality of infants, we must analyse the various mortality statistics into their different constituents and ascertain how the mortality rates are altered by the elimination of one or other of these factors.

A comparison of the statistics of infant and general mortality rates in England and Wales between 1841 and 1925 (Table XLII) reveals two instructive facts, viz., (*a*) between 1841 and 1870 both these rates were practically stationary at about 150 per 1,000 births and about 21·5 per 1,000 population respectively; (*b*) after the introduction of general sanitary improvements in 1872 and 1875 both rates began to fall, but whilst the general death-rate has been steadily falling from that time onwards, the infant mortality rate fluctuated till the beginning of this century, when the regular steady decline began. Hence we conclude that (*a*) to some extent infant mortality is affected by general sanitary conditions, but that (*b*) in addition to some common factor or factors there must also be certain circumstances which are responsible for a high infant mortality but which have no influence upon the general death-rate. Or it is possible that the effect of the improved sanitation upon infant mortality was counterbalanced by some other unfavourable circumstances. Similar conclusions can be drawn from a comparison of the relative mortality statistics for the first year of life and for each of the subsequent four years.

Let us now split up the statistics relating to the first year of life into (*a*) deaths occurring in early infancy, and (*b*) those occurring in later infancy (see Table XXXVII and Charts, figs. 30 and 31). Such an analysis reveals the facts that:—

(1) The decline, since 1902, in death-rate of infants under 3 months is less than that in the case of infants between 3 months and 1 year.

(2) The decline during the first 3 months is almost entirely due to a fall in the death-rate between 1 and 3 months (i.e., a fall of 56 per cent. between 1902 and 1923), the mortality in the first month having been comparatively slight (viz., 22 per cent.) during the same period.

The questions that at once arise are: (i) Why is the early infant mortality so proportionately high? (ii) Why is its rate of decline so proportionately low? (iii) Can we accelerate the rate of decline in each case, and if so, to what limit?

It is here that an elementary knowledge of the principles of infant physiology will help us to solve our problems.

Physiological Peculiarities of Infant Life.—The infant at birth is like a fish out of water. Inside its mother's womb it lived immersed in a water bath of constant body temperature, where microbes were absent, and where, in addition to being protected from all sorts of injury or other stimuli—tactile, thermal, auditory, &c.—it received its nourishment as well as its oxygen direct into its blood without any effort on the part of its digestive or respiratory systems. During the process of birth the fœtus moulds itself to the size of the maternal passages in the same way as a cork moulds itself to the size of a bottle's neck. The effects of this pressure make themselves felt on the whole of the fœtus, but especially on the head, so that in unfavourable cases the child may be born dead or die during its early stages of infancy, or may grow up physically or mentally maimed if it manages to escape with its life.

TABLE XLIII.—NUMBER OF DEATHS IN EACH PART OF INFANCY AND DURING THE FOLLOWING FOUR YEARS OF LIFE IN ENGLAND AND WALES IN 1921.

Age at death	Number of deaths		Death-rate per 1,000 births		Percentage of total infant mortality	
Under 1 day	9,150		10·77		13·01	
1-7 days	9,873		11·63		14·05	
1-4 weeks..	10,909		12·85		15·52	
Total under 4 weeks		29,932		35·25		42·58
4 weeks to 3 months	12,598		14·84		17·93	
Total under 3 months		42,530		50·09		60·51
3-6 months	11,897		14·02		16·94	
6-9 months	8,544		10·07		12·17	
9-12 months	7,279		8·58		10·37	
Total under 1 year		70,250		82·76		100
1-2 years	15,636					
2-3 years	4,994					
3-4 years	3,177					
4-5 years	2,584					
Total under 5 years		96,641				
Deaths at all ages		458,629				

As soon as it has successfully accomplished its hazardous journey and passed safely through the *Arc de Triomphe*, as the maternal pelvic arch may rightly be called, the infant finds itself in entirely new surroundings. It is now thrown upon its own

resources in a strange and rigorous microbe-laden atmosphere of ever-changing temperature and purity. It must not only recover from the injuries received during birth, but must fight its own battles to keep itself alive. It has to keep itself warm by means of its own heat-regulating mechanism, it must feed itself by means of its own digestive apparatus, and inhale oxygen by means of its own respiratory mechanism; its circulatory system, especially its heart, has to go through certain structural changes to adapt itself to its new mode of breathing, and in addition the infant has to accommodate itself to various sensory stimuli such as heat, light, sound, &c., to which it is quite unaccustomed. No wonder, therefore, that nearly half of the total mortality of infants under 1 year occurs during the first month of life, and that about one-third of the entire neo-natal mortality takes place on the first day after birth (see Tables XLIII-XLV).

TABLE XLIV.—THE DEATH OF INFANTS ACCORDING TO AGE AT DEATH. (United States Registration Area for Births, 1919.)

Age at death	Infant deaths per 1,000 births	
Under 1 day	14·5	Under 1 week = 28·7
1 day	4·5	
2 days	3·4	
3-6 days	6·3	
1 week	5·9	Under 1 month = 41·5
2 weeks	3·8	
3 ,, ...	3·1	
1 month	7·3	Under 1 year = 86·6
2 months	5·9	
3-5 months	13·7	
6-8 ,, ...	10·3	
9-11 ,, ...	7·9	

The following figures give the mortality rates of children at the beginning of this century in France:—

TABLE XLV.

Out of 1,000 infants born alive, there died

During the first five days	17
,, ,, ,, month	46
,, ,, ,, year	160
,, ,, ,, two years	210
,, ,, ,, three ,, ...	235
,, ,, ,, four ,, ...	252
,, ,, ,, five ,, ...	265
,, ,, ,, ten ,, ...	321
,, ,, ,, fifteen ,, ...	355

The general mortality at all ages was twenty out of every 1,000 living persons; whilst the death-rate at ages from 40 onwards was only eleven per 1.000 living. Indeed, the mortality in early infancy was about equal to that of people over 80 years old.

Many writers, impressed by the very slow decline in the **neo-natal mortality**, have come to the conclusion that such deaths are not preventable and are due to pre-natal hereditary causes over which we have no control. I do not think, however, that this gloomy view is justified. Injury at birth and syphilis are responsible for a certain—though small—number of neo-natal deaths, which, as we saw in the last chapter, ought to be entirely eliminated by improved obstetric technique and

TABLE XLVI.—DEATH-RATE PER 1,000 BIRTHS.

During first week after birth		During first month after birth	
Dewsbury	41·4	Workington ..	61·0
Batley	37·5	Blyth	58·0
Bradford	30·7	Todmorden ..	54·7
Stepney	20·3	Doncaster	31·0
Leyton	17·8	Penge	27·2
Hornsey	16·2	Watford	25·9

TABLE XLVII.—INFANT MORTALITY IN DIFFERENT PARTS OF ENGLAND AND WALES, 1923.

Area	Under 4 wks.	4 wks.-3 mths.	3-6 mths.	6-9 mths.	9-12 mths.	Total under 1 year
All areas (all infants)	31·88	11·33	10 02	8·39	7·74	69·36
All County Boroughs	34·10	10·05	12·29	10·59	9·90	79·93
All Rural Districts	31·97	9·80	7·38	5·99	5·60	60·74
Northern County Boroughs ..	37·18	14·18	14·04	12·80	11·80	90·00
Southern Rural Districts ..	27·15	8·11	4 81	4·39	4·19	48·59
London	25·93	9·61	11·11	7·64	6·97	61·26
Legitimate—						
County Boroughs	33·02	12·35	11·62	10·19	9·60	76·78
Rural Districts	30·82	9·21	6 98	5·86	5·52	58·39
Illegitimate—						
County Boroughs	58·94	29·51	28·04	20·01	16·88	153·38
Rural Districts	55·50	22·00	15·47	8·80	7·24	109 01

The fact that infant mortality is less in rural than in urban districts was known to Captain John Graunt of London, in 1662, but the real reason for this phenomenon is not understood even now.

proper pre-natal supervision. Diarrhœa, and to a certain extent pneumonia and bronchitis, are preventable—as we shall see presently. A certain considerable proportion of the *cases* of prematurity can, as I also showed in the last chapter, be prevented by skilled ante-natal treatment (e.g., syphilis, toxæmia, tuberculosis, &c.), and their *deaths* prevented by patient, pains-taking and intelligent post-natal care, viz., proper feeding and

protection against exposure, &c. Even a certain proportion of the deaths coming under the heading of congenital malformations can be prevented, not indeed by diminishing their incidence by pre-natal care, but by diminishing their fatality rate by proper post-natal attention and treatment (e.g., cleft palate, certain cases of congenital heart disease, &c.). It is only the small group of those congenital malformations which are compatible with fœtal life but are incompatible with post-natal life, such as obliteration

TABLE XLVIII.—ENGLAND AND WALES. INFANT MORTALITY BY SEX, AGE AND LEGITIMACY, 1923.

	Deaths per 1,000 births								
	All infants			Legitimate infants			Illegitimate infants		
	M.	F.	P.	M.	F.	P.	M.	F.	P.
All causes—									
Under 4 weeks ..	36	28	32	35	26	31	61	51	56
1-3 months	13	9	11	12	9	11	29	23	26
3-6 ,, ...	11	9	10	11	8	10	25	19	22
6-9 ,, ...	9	7	8	9	7	8	15	15	15
9-12 ,, ...	9	7	8	8	7	8	13	11	12
Total under 1 year	78	60	69	75	57	68	143	119	137
All ages under 1 year—									
Infectious diseases..	4·45	4·02	4·24	4·37	3·95	4·16	6·43	5·67	6·06
Tuberculosis ..	1·56	1·17	1·36	1·51	1·15	1·32	2·71	1·63	2·19
Diarrhœa, &c. ..	7·81	5·78	6·82	7·41	5·39	6·42	17·01	14·85	15·96
Developmental and wasting	33·43	25·89	29·74	32·48	24·98	28·83	54·78	47·29	51·13
Miscellaneous ..	31·05	23·16	27·20	29·66	22·01	25·92	63·22	49·38	56·47

M = male; F = female; P = persons.

Hence in comparing infant mortalities of two places for the purpose of a campaign, to wipe out the differences and to reduce all the mortalities to the level of the lowest, we must take account not only of such **uncontrollable** environmental and racial differences as climate and soil, and racial composition of population, &c., but also of the relative proportions of legitimate and illegitimate infants, male and female infants, and of their age distribution in the two places.

of the œsophagus, or of the bile-ducts, or of the urinary passages, &c., which are not amenable to preventive or curative treatment. Indeed, not only does a closer examination of statistics of neo-natal deaths in different areas confirm this conclusion (Table XLVI), since it shows that such mortality rates vary in different places from 26 and 61 per 1,000, and first-week mortality rates vary between 16 and 41 per 1,000, but that there is a co-variation, or correlation of variability between neo-natal and later infant mortality

rates, so that in places where the one is high the other also tends to be high, and vice versa. Moreover, the great variability of the neo-natal mortality rate with legitimacy and sex (Table XLVIII) is further evidence of its preventability. But the best proof of all of the preventability of early infantile deaths is the fact that they have actually been prevented. Dr. Dublin has analysed the results of intensive pre-natal and early post-natal care—other than the provision of skilful midwifery attendance and of proper antisyphilitic treatment—given by the Maternity Centre Association of New York to 8,743 patients, and his figures are given in Table XLIX. It will be seen that congenital malformations, prematurity and diarrhœa deaths have been greatly diminished by such supervision. From the character of the supervision, no reduction could be expected in the mortality rates from syphilis and birth injuries. **Hence we may conclude that neo-natal mortality can and ought to be prevented to a very large extent.**

TABLE XLIX.—NUMBER OF DEATHS UNDER ONE MONTH FOR CERTAIN DISEASES, PER CENT. OF TOTAL, AND DEATH-RATE PER 1,000 LIVE BIRTHS, NEW YORK CITY, 1920, AND EXPERIENCE OF MATERNITY CENTRE ASSOCIATION, NEW YORK CITY, 1919-21.

Causes	New York City, 1920			Maternity Centre Association, New York City, 1919-21.		
	Number of deaths	Per cent. of total	Mortality per 1,000 births	Number of deaths	Per cent. of total	Mortality per 1,000 births
Total:	4,651	100	35·0	207	100	25·9
Syphilis	63	1·4	0·5	7	3·4	0 9
Convulsions	14	0·3	0·1	6	2·9	0·8
Bronchitis and pneumonia	352	7·6	2·7	18	8·7	2·3
Diarrhœa	201	4·3	1·5	1	0·5	0·1
Congenital malformations	471	10·1	3·5	13	6·3	1·6
Prematurity	1,957	42·1	14·7	38	18·4	4·8
Congenital debility ..	667	14·3	5·0	55	26·6	6·9
Injuries at birth ..	627	13·5	4·7	32	15·5	4·0
Other causes	299	6·4	2·3	37	17·9	4·6

The reason that the Notification of Births Act (due to Dr. S. G. Moore and Mr. Benjamin Broadbent, of Huddersfield) and Midwives Act have so far not given as good results as could be expected, is probably to be explained by the fact that the objects of the Acts have not yet been satisfactorily carried out.

In the first instance the Notification of Births Act of 1907 was permissive, and it was only in 1915 that the Notification of Births (Extension) Act made the notification of births in this country compulsory. Secondly, even the 1915 Act is so worded as to render evasion of it, by the unremunerated and occasionally unwilling doctor or midwife, comparatively easy. For although failure to notify is punishable by a fine of 20s., the Act, which imposes the duty of notification upon the father as well as upon the doctor or midwife, states that the delinquent shall not be liable to a fine if he or she had reasonable grounds for believing that birth had already been notified by some other person.

TABLE L.—STATISTICAL CONSTANTS FOR FORTY-SEVEN AMERICAN CITILS, 1920.

Constant	Neo-natal mortality	Mortality of age period 1—12 months
Mean	43·97 ± 3·51	49·75 ± 10·01
Range	20·3	64·2
Standard deviation	5·20 ± 0·362	14·84 ± 1·032
Coefficient of variability ..	11·826 ± 0·834	29·829 ± 2·252
Coefficient of co-variation ..	0·4580 ± 0·0777	
Coefficient of regression ..	0·161 ± 0·031	1·306 ± 0·249

The statistical constants calculated by Dublin are of considerable interest (see Table L). The coefficient of variability (which is the ratio, per cent., between the standard deviation and the mean, viz., $\frac{\text{S.D.}}{\text{mean}} \times 100$) is nearly three times as great for the ages 1 to 12 months as for the first month. This is not surprising if we take the time element into consideration, since there is very much more room for the play of environmental factors during eleven months than during one month.

Hence we have an explanation of the reason why neo-natal mortality is so high, and why its decline has been very slow.

We now have to investigate **which of the pathological causes of death have shown the greatest reduction in the mortality rate for the ages 1-12 months ?** An answer to this question will help us to gauge the relative importance of the various factors in the hygiene of infancy and will point the way to future methods of intensive infant welfare work. Further knowledge of the physiological peculiarities of infancy, as well as examination of

the trend of mortality rates from the various principal certified causes of infant deaths, will help us to solve this problem.

Second Important Peculiarity of Infancy.—The infant's digestive apparatus is immature and incomplete. The various digestive glands are not yet fully developed and the muscular walls of the digestive tract are feeble. Hence we should expect to find **diseases of the digestive system** to be responsible for a large percentage of the total infant mortality. This is borne out by statistics, but we also find that with advance of knowledge of the infant's physiological requirements, the mortality from this cause is rapidly declining (see Tables LI-LVI).

TABLE LI.—DEATHS PER 1,000 BIRTHS OF INFANTS UNDER 1 YEAR FROM VARIOUS CAUSES FOR THE YEARS 1905 AND 1923 (ENGLAND AND WALES).

Cause of death	1905	1923	Reduction per cent.
Diarrhœa and enteritis	21·47	6·82	68·2
Bronchitis and pneumonia.. ..	21·30	13·43	36·9
Infectious diseases	7·80	4·24	45·7
Convulsions	13·84	4·18	69·8
Tuberculous diseases	5·14	1·36	73·5
Prematurity	20·30	17·65	13·1
Suffocation (including overlaying)..	1·77	0·55	68·9
Congenital malformations	4·75	4·16	10·3
Syphilis	1·32	1·05	20·5
All causes	128	69	46

TABLE LII.—DECLINE OF INFANT MORTALITY FROM DIFFERENT CAUSES OF DEATH.

Diseases	1891-1900	1901-1910	1911-1915	1916-1920	1920-1921	Percentage difference between 1920-21 and 1891-1900
Common infectious diseases ..	10·03	8·09	7·42	5·08	3·91	61·0
Tuberculosis ..	7·85	5·17	3·08	2·00	1·50	80·4
Diarrhœa, &c. ..	25·17	20·47	19·24	9·34	10·88	56·7
Bronchitis and pneumonia ..	13·06	10·68	9·60	9·30	8·04	38·4
Rickets	0·55	0·57	0·39	0·32	—	—
Overlying	2·05	1·75	1·40	0·83	0·60	70·7
Convulsions ..	18·23	14·14	8·41	6·79	—	—

TABLE LIII.—TOTAL INFANT MORTALITY AND MORTALITY DUE TO DIARRHŒAL DISEASES IN INFANTS UNDER 1 YEAR OLD, IN ENGLAND AND WALES, BETWEEN 1883 AND 1923.

Year	Total infant mortality	Mortality due to diarrhœa
1883	137	12
1888	136	11
1893	159	28
1898	160	37
1903	132	18
1908	120	20
1913	108	19
1918	97	10
1923	69	4

TABLE LIV.—DEATHS FROM VARIOUS CAUSES UNDER 1 YEAR, 1905-1923 (ENGLAND AND WALES).

Causes	1905	1908	1911	1914	1915	1916	1917	1918	1919	1920	1921	1922	1923
All causes ..	128	120	130	105	110	91	96	97	89	80	83	77	69
Diarrhœa, &c.	21	20	36	17	15	11	10	10	9	8	14	9	7
Bronchitis and pneumonia	21	20	18	18	23	17	20	20	19	17	15	18	13
Infectious ..	8	7	8	7	9	5	6	8	3	4	4	5	4
Convulsions	14	13	10	8	9	8	8	7	6	6	5	5	4
Developmental and wasting	44	42	41	38	37	36	37	35	37	32	33	31	30

TABLE LV.—(1) DEATHS UNDER 1 YEAR FROM VARIOUS CAUSES FOR THE YEARS 1911 AND 1920 IN THE U.S. REGISTRATION AREAS.

Causes of deaths	1911		1920		Reduction per cent.
	Per 1,000 births	Per cent. of total	Per 1,000 births	Per cent. of total	
All causes	112·9	100	96·7	100	14·3
Diseases of digestive system (diarrhœa, &c.)	32·3	28·6	19·4	20·1	40
Diseases of respiratory system (bronchitis, pneumonia, &c.)	17·0	15·1	15·0	15·5	11·8
Communicable diseases (pertussis, measles, &c.)	10·6	9·4	12·0	12·5	— 13·2
Diseases of early infancy (congenital debility, &c.)	41·3	36·6	40·6	42·0	1·7
Other causes	11·6	10·3	9·6	9·9	17·2

TABLE LVI.—(2) PERCENTAGE OF REDUCTION IN MORTALITY IN NEW YORK FROM EACH OF THE IMPORTANT CAUSES OF DEATH OF INFANTS IN 1919 ON THE BASIS OF CONDITIONS PREVAILING IN 1885.

Cause of death	Mortality rate per 1,000 births		Per cent. reduction
	1885	1919	
All causes	273·6	81·6	70·2
Diarrhœa, &c.	84·3	15·9	82·3
Respiratory	42·2	15 5	63·3
Communicable diseases	20·8	1·9	90·9
Congenital	42·1	37·2	11·6
Other causes	84·2	11·2	86·7

From these tables we see that the reduction has been mainly in the case of diarrhœa and convulsions—diseases caused by unsuitable or infected food. Hence we have two indications for preventing infant deaths, viz. :—

(*a*) Improvement in infant feeding.

(*b*) Improvement in dairy and domestic sanitation to avoid contamination of milk.

Under the first heading we must concentrate upon the encouragement of breast-feeding, since statistics (see Tables LVII-LIX) conclusively prove that at all ages a breast-fed baby has a much better chance to live than a bottle baby. This is not only because breast milk contains the various ingredients in the proper physiological proportions required by the infant in the most digestible form, but also because the mother's milk, coming direct from the breast into the child's mouth, is free from harmful

TABLE LVII.—RELATIVE MORTALITIES FROM VARIOUS CAUSES AMONGST BREAST-FED AND ARTIFICIALLY-FED INFANTS (Woodbury).

Cause of death	Deaths among artificially-fed infants		
	Actual	Expected	Ratio of actual and expected
Total	1,047	268·4	3·9
Gastro-intestinal	495	64·2	7·7
Respiratory	185	100·4	1·8
Malformations	26	5·9	4·4
Early infancy	165	27·1	6·1
Epidemic diseases	74	32·7	2·3
External causes	3	2·5	1·2
Unknown	18	3·7	4·9
Other causes	81	31·9	2·5

organisms. In addition, breast milk also probably transfers immunizing substances from the mother to the baby, thus conferring upon it a certain degree of immunity against infectious diseases, and it also contains those vitamins which protect the infant against rickets and scurvy.

It has been found that the correlation coefficient between artificial feeding and infant mortality is 0·765 ± 0·029 (Greenwood and Brown).

TABLE LVIII.

Infant Mortality and Type of Feeding (other circumstances being equal). Total number investigated = 22,422.

Month of life	Monthly death-rate for each month of life per 1,000 infants				
	All types of feeding	Exclusively breast-fed	Partly breast-fed	Exclusively artificially-fed	Ratio of bottle-fed and breast-fed
First ..	21·5	16·9	36·4	54·7	(3 : 1)
Second ..	9·3	5·8	14·7	24·6	(4 : 1)
Third ..	8·1	3·7	12·9	21·2	(6 : 1)
Fourth ..	8·0	3·4	9·0	19·2	(5 : 1)
Fifth ..	7·7	3·3	5·7	18·1	(6 : 1)
Sixth ..	7·4	2·1	5·9	17·7	(8 : 1)
Seventh ..	6·3	1·9	4·0	14·1	(7 : 1)
Eighth ..	5·8	2·9	3·3	11·3	(4 : 1)
Ninth ..	5·7	3·2	2·9	10·7	(3 : 1)
Tenth ..	5·3	3 8	2·3	9·3	(2 : 1)
Eleventh ..	3 9	2·4	2 5	6·0	(2 : 1)
Twelfth ..	4·5	4·4	2·7	6·4	(1·5 : 1)

The ratios in brackets in the last column represent at each month the proportionate number of deaths in the infants exclusively bottle-fed and those exclusively breast-fed.

TABLE LIX.—INFANT MORTALITY AND TYPE OF FEEDING.

Month of life	Infants surviving at beginning of specified month of life						
	Total	Exclusively breast-fed during the month		Partly breast-fed during the month		Exclusively artificially-fed during the month	
		Number	Per cent.	Number	Per cent.	Number	Per cent.
First ..	22,422	19,438	86·7	686	3·1	2,287	10·2
Second ..	21,939	17,178	78·3	1,294	5·9	3,457	15·8
Third ..	21,735	15,509	71·4	1,786	8·2	4,431	20·4
Fourth ..	21,560	13,516	62·7	2,668	12·4	5,366	24·9
Fifth ..	21,387	12,263	57·3	3,317	15·5	5,797	27·1
Sixth ..	21,222	10,969	51·7	4,098	19·3	6,145	29·0
Seventh ..	21,066	8,641	41·0	5,801	27·5	6,615	31·4
Eighth ..	20,934	7,343	35·1	6,671	31·9	6,911	33·0
Ninth ..	20,812	5,853	28·1	7,684	36·9	7,268	34·9
Tenth ..	20,693	4,248	20·5	8,591	41·5	7,845	37·9
Eleventh ..	20,584	3,278	15·9	9,062	44·0	8,235	40·0
Twelfth ..	20,504	2,736	13·3	9,145	44·6	8,616	42·0

The above statistics, presented by Dr. Woodbury (Director, Statistical Research Children's Bureau, U.S. Department of Labour, and published in the *American Journal of Hygiene*, November, 1922), effectively dispose of the contention of some (e.g., Karl Pearson and Mary N. Karn) to the effect that the increased mortality amongst artificially-fed babies is not due to the feeding but to some hereditary circumstances which made artificial feeding necessary, viz., congenital debility due to prematurity, twin birth, or poor health of mother, &c., since all these circumstances have been taken into account by Woodbury in the compilation of his statistics. For when premature infants, or plural births, &c., are excluded, the ratio between the mortality of bottle-fed and that of breast-fed infants is reduced only from 3·9 to 3·3!

Dr. Woodbury's statistics also show that the type of feeding has a cumulative effect during the early months of life, so that the longer the infant is breast-fed during the early months the better its chance of survival after weaning.

Hope, of Liverpool, showed that in 1,000 breast-fed babies under three months there were 20 deaths from diarrhœa; in 1,000 bottle-fed babies under three months there were 300 deaths.

Newsholme gives 10 per cent. for breast-fed and 90 per cent. for bottled-fed.

It is not artificial feeding *per se*, since among the well-to-do bottle babies do well; the secondary factors are bad milk, unsuitable food, improper methods of feeding, lack of maternal care, bad surroundings, heat, humidity, dirt, overcrowding, which favour spread of infection.

The following are French statistics on the same subject.[1] In 1920 7,167 infants of less than 1 year attended the 180 infant consultations of the department of Seine-et-Oise. 5,091 of these were breast-fed—of these 117 died; mortality = 22·9 per 1,000. 2,076 were bottle-fed—of these 225 died; mortality = 108·3 per 1,000. Total dead = 342, giving the average mortality of both classes as 47·7 per 1,000.

The general infant mortality in that department during 1920 was 72·2 per 1,000 (but see p. 181).

Under the second heading of "Prevention of Mortality from Gastro-intestinal Disease," come the following:—

(i) Healthy and clean milk supply of low bacterial content (see Chapter XVI, p. 407, *et seq.*).

[1] "Rapport de la fédération départementale des Œuvres d'assistance maternelle infantile de Seine-et-Oise," 1920.

(ii) Domestic cleanliness and sanitation to ensure non-contamination of the milk at home.

(iii) The destruction of flies—*by eliminating their breeding places, especially horse manure, which is responsible for some 95 per cent. of the house-fly population of a community*—as there is no doubt that a large proportion of infant intestinal disorders is brought about by milk contaminated by flies. The nursery windows should have gauze screens to keep out flies.

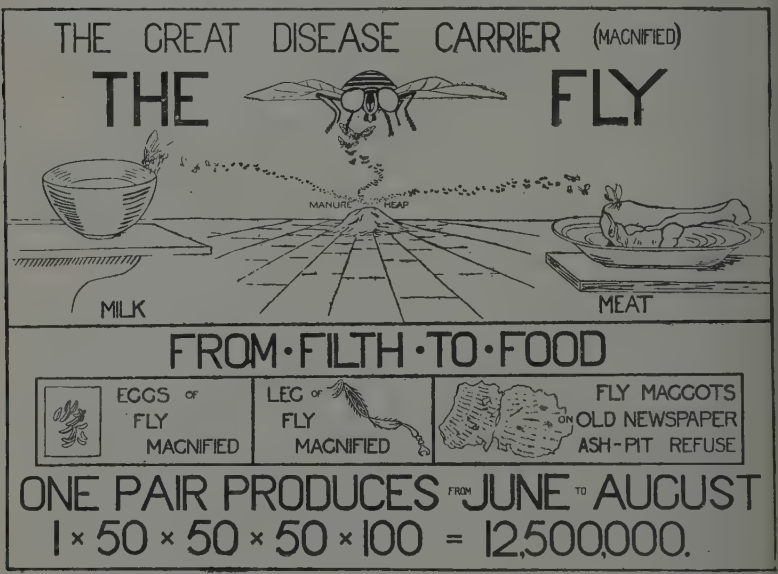

Fig. 32.

(iv) Instruction of the mother in the proper method of preparing and giving the food to her infant. She must be taught how to store the milk, how to modify it to suit the requirements of her baby, and how to keep the feeding-bottle, &c., scrupulously clean.

It is confidently to be expected that diarrhœa and other troubles arising from faulty feeding will gradually be eliminated as a cause of infant mortality. In 1919 the University of Minnesota instituted an intensive campaign in favour of breast-feeding in Minneapolis, with the result that an average infant mortality

of 82 for the previous five years was reduced to 65 in 1919 In New Zealand the mortality of infants under 2 years from diarrhœa was 3·5 per 1,000, and for Dunedin City it was only 0·8 per 4,000 for the years 1918-22, whilst in 1923 there was no death from diarrhœa in Dunedin at all. Dunedin is the seat of the most intensive education of mothers.

Artificial Feeding and Infant Diarrhœa.—It had long been noticed that infant diarrhœa was more prevalent in artificially-fed babies than in those fed on the breast, especially in the summer months. Hence a relationship must exist between cow's milk and the prevalence of the disease as well as between the latter and the atmospheric temperature. It was therefore reasonable to assume that the temperature had an effect upon the milk which rendered it poisonous to infants. Moreover, it was noticed that children on dairy farms drank cow's milk with impunity, whilst of those in the city who drank the milk from one to three days later, a large percentage died from diarrhœa. Moreover, the longer the interval between milking and feeding the greater was the mortality from diarrhœa.

The relationship between cow's milk and diarrhœa is not determined by the chemical composition of the milk, i.e., by the percentage of carbohydrates, fats, and proteins, but by its bacterial content. Milk drawn through sterilised capillary tubes inserted into the udders of healthy cows is sterile, but milk drawn from the udder in the ordinary way, even under the most stringent conditions, contains bacteria of all sorts.

Summer Diarrhœa (or Epidemic Diarrhœa).—The exact bacteriology and pathology of this disease are as yet unknown. What is known with certainty is that diarrhœa occurs during the summer months, is commonest and most fatal in children under 2 years of age, especially between the ages of 3 and 6 months (in infants that are artificially fed), and that since the beginning of this century the annual mortality rate from this disease has been gradually declining, especially since 1911.

Relation between Summer Diarrhœa and Air and Soil Temperatures.—Epidemic diarrhœa is commonest in hot summers (e.g., 1911) and least common in cool summers (e.g., 1912). That the temperature of the air is not the direct cause of the disease is shown by the facts that: (1) There is no resemblance between the mortality curve and the curve of the mean weekly maximum air temperature; (2) the curves are not contemporaneous, but the mortality curve lags several weeks behind the temperature

curve (see next paragraph). Similarly, it has been shown that the view of Ballard and others to the effect that high soil temperature is the cause of the disease is not borne out by measurements with the 4-ft. earth thermometer.

Relation between Summer Diarrhœa and Flies.—There is a close relationship between the mortality curve of the disease and the curve of the annual prevalence of flies, and as the breeding of flies is favoured by high atmospheric temperature, the latter may indirectly influence the prevalence of the disease. This would account for the *lagging* of the mortality curve behind the temperature curve, since it takes some time for the fully-developed flies to escape. Hence we can say that there is a close association between summer diarrhœa and flies.

Manner in which the Flies Convey the Disease.—The flies convey the infecting organism from fæces to the food (especially the milk and sugar) of the healthy infant (see fig. 32). This is shown by the fact that in some outbreaks in which a definite infecting organism (Morgan's No. 1 bacillus) could be isolated from the patient's fæces, the same organism was found in the intestines of the flies associated with those outbreaks, but not in the intestines of flies from non-infected neighbourhoods. Further, it has been shown that the milk is not infected at the *source*, since there is no correlation between the incidence of the disease and the source of the milk supply. Hence the contamination takes place in the home, and, indeed, the dirtier the house and the greater, therefore, the opportunity for fly-breeding, the greater the incidence of summer diarrhœa. Lastly, when contamination of milk by flies is prevented, diarrhœa disappears.

The Organism Associated with the Disease.—Numerous organisms have been found in the intestinal discharges of infants suffering from the disease, e.g., the dysentery bacillus, especially of the "Flexner-Harris" and "Shiga" types (in 60-70 per cent. of the cases in the United States), the *Bacillus fæcalis alcaligenes* (in Australia), the typhoid groups (in Manchester), *B. coli*, and other organisms, but the one most commonly found associated with the disease in England is Morgan's No. 1 bacillus.

The occasional occurrence of the disease in breast-fed babies is probably due to lack of cleanliness on the part of the mother, who may have infected her fingers with the *B. coli* of her own intestines and failed to wash her hands before giving the baby the breast.

Prevention.—The following may be called the ten commandments of the prevention of summer diarrhœa :—

(1) During the summer months the milk should be scalded immediately it is delivered.

(2) Milk and sugar must be kept covered, and the milk must be kept in a cool place, preferably in a refrigerator. A simple way of keeping milk cool at home is shown in fig. 33.

(3) Feeding-bottles and teats must be kept scrupulously clean.

(4) No comforter should be allowed.

(5) Before preparing the baby's food the mother or nurse should carefully wash her hands with hot water and soap.

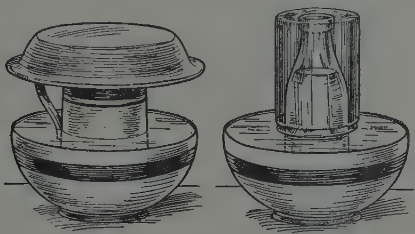

FIG. 33.—Right way to keep milk in the home (cool and covered). The bowl contains cold water.

(6) The mother and nurse should, without fail, wash their hands carefully and scrub their finger-nails (which should be kept short) after changing the baby's napkins or attending to the stools of other children. Fingers should not be put into the baby's mouth to feel for teeth, or for any other reason, unless they have been carefully washed.

(7) Infants or young children should not be allowed to crawl on the floor, lest their hands and clothes become dirty and the infected part is carried to the mouth.

(8) Flies must be kept away from the intestinal discharge by:—

(*a*) Putting soiled napkins into enough water to cover them. E. C. Levy, of Richmond, Virginia, greatly reduced infant mortality by this one procedure alone (see fig. 34).

(*b*) Keeping the w.c. pans well flushed and clean.

(*c*) Keeping the house and immediate surroundings free from excreta and other refuse, in this way preventing the breeding of flies.

(*d*) The streets ought to be kept free from fæcal matter, or other dirt or manure.

(9) The most important thing of all is that baby should be breast-fed under reasonably clean conditions.

(10) Lastly, no breast baby should be weaned during the hot months, July-September.

Pearl and Ilsley,[1] taking illiteracy as a measure of stupidity and ignorance, find the net correlation coefficient between mortality from diarrhœa and ignorance to be as high as + 0·715 ± 0·015.

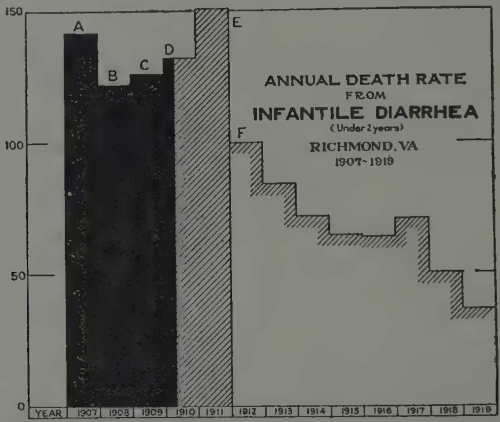

FIG. 34.—Influence of diapers in spreading diarrhœa. At A, inspection of dairies was introduced. At B, the entire milk supply was put in "good shape." At C, all the privies were made sanitary. At D, health visiting was introduced. At E, the rôle played by infection was recognised. At F, instruction given in care of diapers, and since then decline in mortality rate became progressive.

The importance of cow's milk in infant feeding is shown by the following figures :—

Percentage of breast-feeding among all children = 75 per cent.

Percentage of breast-feeding among those that die of diarrhœa = 22 per cent.

So that whilst the ordinary proportion of breast to bottle in all children = 3 : 1, the proportion in the case of diarrhœa babies = about 1 : 4. In other words, bottle-feeding is twelve times as common amongst the diarrhœa babies as amongst all babies.

Further evidence of the superiority of natural over artificial feeding of infants is found in a comparison of the infant mortality rates of such countries as Ireland and Norway, where

[1] *Amer. Journ. Hyg.*, ii, 1922, p. 587.

breast-feeding is the rule, with Germany and Austria, where bottle-feeding is largely resorted to.

The infant mortality rates in the latter are about two and a half times as high as in the former. France is intermediate, both in regard to the prevalence of breast-feeding and infant mortality.

Certain investigations have shown that even those bottle-fed babies which escape death grow up less vigorous, shorter, more susceptible to digestive, infectious, tuberculous and other troubles than those fed on the breast. Rickets is, of course, much more prevalent amongst them.

Causes of Prevalence of Artificial Feeding.—Granted that breast-feeding is the greatest safeguard against infant mortality, it is of interest to inquire why many women resort to the artificial substitute. The causes may be classified as follows:—

(1) *Physiological.*—There are some women of neurotic temperament who do not secrete sufficient milk (see Chapter XIV, p. 372).

(2) *Pathological.*—Women suffering from infectious diseases (other than syphilis), or from severe cachexia, or from deformity of the nipples, cannot feed their babies.

TABLE LX.

Age	Weight in kilos	Area of skin in sq. cm.	Area of skin per kilo body weight
4 days (premature)	1·50	1,266	841
New-born	2·10	1,476	704
15 day's	2·98	2,129	711
3 months	3·52	2,280	648
6 ,, ...	5·14	2.961	576
1 year	9·10	4,800	527
5 years	14 96	6,722	409
10 ,, ...	21·70	9,166	423
15 ,, ...	30·14	11,402	378
Adult	63·65	14,079	221

(3) *Social.*—This constitutes the most important group. Amongst the well-to-do it is the selfishness of the mother and her desire for enjoyment away from home that is the chief obstacle against breast-feeding. This is often encouraged by the doctor or midwife. Amongst the poorer classes it is mostly the necessity to go out and earn her livelihood that forms the chief obstacle, but in some cases it is ignorance of the consequences that is the cause.

It is the duty of physicians, nurses and sociologists to remedy the first and to fight against the third of these groups of causes.

The other peculiarities of infancy are: (1) The relatively large surface area of its body (see Table LX), so that it loses more heat in proportion to its bulk than does an older person. (2) Its heat regulating mechanism is defective, so that it cannot control its internal temperature with changes in the temperature of its surroundings. Hence it must be properly covered to protect it from cold, and it must not be subjected to sudden changes of temperature. This applies more especially to premature infants.

TABLE LXI.—PROPORTIONATE MORTALITY, AGES 1-5, U.S. REGISTRATION AREA, 1910-1915. (Whipple.)

Age 0-1		Age 1-2		Age 2-3		Age 3-4		Age 4-5	
	%		%		%		%		%
Congenital debility	25·9	Diarrhœa and enteritis	27·3	Diarrhœa and enteritis	13·2	Diphtheria	17·0	Diphtheria	18·8
Diarrhœa and enteritis	24·4	Broncho-pneumonia	14·4	Diphtheria	12·2	Broncho-pneumonia	7·8	Scarlet fever	7·6
Broncho-pneumonia	8·1	Pneumonia	9·5	Broncho-pneumonia	11·1	Pneumonia	7·6	Pneumonia	6·8
Other diseases	11·8	Diphtheria	5·8	Pneumonia	8·9	Diarrhœa and enteritis	7·3	Broncho-pneumonia	6·0
		Measles	5·9	Measles..	5·5	Scarlet fever	7·0	Burns ..	5 7
		Whooping cough	4·6	Scarlet fever	4·7	Burns ..	5·9	Diarrhœa and enteritis	5 0
		Tuberculous meningitis	3·1	Whooping cough	4·0	Measles..	4·4	Tuberculous meningitis	3·8
				Tuberculous meningitis	3·9	Tuberculous meningitis	4·0	Measles..	3·5
						Whooping cough	3·2		

(3) The infant's respiratory apparatus is such as to render it particularly liable to congestion. Its nose is narrow, shallow and vascular, its thorax is soft and weak, and its lungs are very vascular. Hence we find that a very considerable proportion of the total infant mortality (between one-sixth and one-fifth) is due to diseases of the respiratory system. Indeed, the mortality would probably be still greater but for the fact that the infant's right heart, which pumps blood into and out of the lungs, is relatively stronger than at later ages, thus helping to compensate to some extent for the disadvantages arising out of

its vulnerable respiratory apparatus. Moreover, during early infancy there are still some immune bodies in its blood—derived from the maternal circulation before birth—rendering it less susceptible to such diseases, and, in the case of breast-fed infants, some immune bodies are absorbed from the milk. It is during the second and third years of life that respiratory diseases assume the greatest importance from the point of view of mortality, as contrasted with digestive troubles which till recently were the most potent cause of death during the first year of life (see Table LXI).

Mortality from Respiratory Diseases.—The tables already presented (LI-LVI) show the very considerable reduction in mortality of infants from diseases of the respiratory system, due chiefly to the diminution in the number of deaths resulting from infectious diseases (principally measles and whooping-cough) of which these diseases are the sequelæ. This gives us a certain indication with respect to prevention, viz., education of parents regarding the danger of exposing young infants to infectious disease, in the foolish and mistaken belief that every child must have these diseases and the sooner they get over them the better. Statistics show that the older the child when it catches the disease the more likely is it to recover. *The prevention of overcrowding* is another means of stopping the spread of communicable diseases. Recent work in Paris has shown that the mode of spread of broncho-pneumonia in baby hospitals is not so much through the air, nor by means of the saliva projected from the baby's mouth in the act of coughing, as by the transmission of the germs conveyed by the nurses' hands from the infants' stools.[1] It has been shown that the air contains very few pneumococci, nor can the saliva be projected farther than about five or six inches from the mouth, but that virulent pneumococci are present in the stools of infants suffering from broncho-pneumonia.

Influence of Climate on Respiratory Diseases.—The next table (LXII) shows that the mortality of infants from respiratory diseases is nearly three times as high in the county boroughs of the north as in the rural districts of the south. The first explanation that suggests itself is that the cold and moist atmosphere of the north is more injurious than the warm and dry atmosphere of the south, but a glance at the third column in the table shows that

[1] Ribadeau-Dumas and Fouet, "La Contagion de Broncho-pneumonies," *Journal de Médecine de Paris*, April 12, 1924.

in the cold and wet atmosphere of the North of Scotland the mortality figures for the various respiratory diseases are practically identical with those of the rural districts of the South of England.

TABLE LXII.—PATHOLOGICAL CAUSES OF INFANT DEATHS IN 1914.

Cause of death	Deaths under 1 year per 1,000 births			
	England and Wales	County boroughs of North	Rural districts of South	Northern Scotland
Total respiratory diseases	25·65	35·03	13·86	15·50
Pneumonia	10·40	14·03	6·01	6·32
Bronchitis	7·75	10·76	4·69	4·86
Whooping-cough ..	4·38	5·31	2·14	2·97
Measles	2·14	3·77	0·27	0·54
Pulmonary phthisis ..	0·35	0·43	0·29	0·18
Other respiratory diseases	0·63	0·73	0·46	0·63
Diarrhœa and enteritis..	17·37	23·54	6·11	6·12
Developmental conditions	35·97	39·42	28·84	26·38
Other diseases	25·63	31·78	16 72	19·08
All causes	104·62	129·77	65·53	67·08

The county boroughs of the North of England have in respect of moisture and temperature the same climate as the northern parts of Scotland, and yet in respect of infant mortality from respiratory diseases the northern boroughs have a death-rate of more than double that of the southern rural districts, whilst the mortality in the latter is practically identical with that in the North of Scotland.

It is probable that dirtiness of the air with regard to smoke contents, together with shutting out of ultra-violet radiation from the sun, is an important cause of high infant mortality from respiratory diseases. It is true that differences in such death-rates occur in different areas of the same town, but it is equally true that different parts of the same town also show marked variations in the amount of solid matter in the air. But when everything is taken into consideration, we must admit that the factors responsible for the mortality of infants from respiratory diseases have not as yet been identified with any degree of certainty. In view, however, of the fact that the respiratory exchanges of the infant are much greater in infancy than in later life, it is clear that there must be some correlation between impurity of the atmosphere and infant mortality. The supply of good hygienic houses, plenty of open spaces, and of garden cities, are measures which ought to be provided by the State in order to diminish the mortality of infants from diseases of the respiratory system.

Seasonal Distribution of Infant Deaths.—The next diagram (fig. 34A) shows that some of the diseases have their greatest incidence and fatality rates at certain months of the year. Hence special attention must be paid to their prevention during those months. During the winter month the infant must be warmly dressed and kept in well-ventilated rooms. But it must not be overdressed, as experience shows that overdressing keeps the child's weight stationary and may also interfere with the proper expansion of the chest and abdomen. Further, it must not be kept too much indoors. The statistics already presented also show what an influence breast-feeding has upon the mortality of infants from respiratory diseases. It is to be remarked about this diagram that the sharp rise in the mortality rate from diarrhœa during the summer months is now in most places a thing of the past.

The Relation between Economic Conditions and Infant Mortality.—Since the time of Malthus (1817) sociologists have associated a high infant mortality with poverty of the parents, and many statistics have been presented which, to the superficial type of mind or the untrained observer, show the correlation between the two (see Table LXIII). But although on philanthropic grounds the relationship certainly ought to be true, yet exact

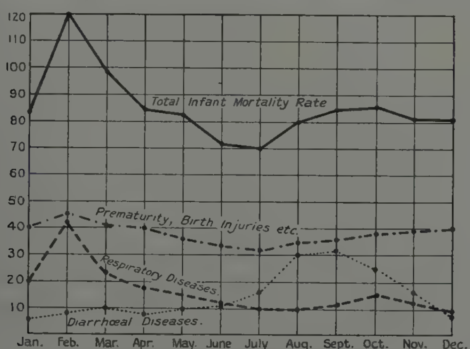

FIG. 34A.

analysis of the statistical data has failed to demonstrate any unambiguous association between poverty and infant mortality. Indeed, the high infant mortality in the mining areas, where wages are comparatively high, and the low infant mortality in Ireland as well as

in the Highlands of Scotland, where wages are very low, show that poverty *per se* cannot be the only cause of the differences in mortality of infants in the various social classes. Greenwood and Brown find that *when other factors are constant*, the correlation coefficient between poverty and infant mortality is utterly insignificant (111 ± 0·071). Most of the recent authorities agree that the general association between excessive infant mortality and poverty is due to the association of the latter with bad housing, low standard of cleanliness, lack of breast-feeding, ignorance, &c.

Relation between Poverty and Infant Mortality.

German Statistics (Groth and Hahn):—

Mean poverty figure ..	1·38	1·88	2·32	2·79	3·65
Mean infant mortality figure	20·9	20·8	26·4	27·4	30·4

Table LXIII.

Infant Mortality by Fathers' Earnings (U.S.A.).

	All earnings	$1,250 and over	Under $550
All cities	111·2	64·3	151·4
Johnstown	130·7	87·6	260·9
Manchester	165·0	58·3	204·2
Brockton	96·7	73·5	67·1
Saginaw	84·6	22·2	192·0
New Bedford..	130·3	59·9	168·7
Waterbury	122·7	68·4	151·1
Akron	85·7	40·0	117·5
Baltimore	103·5	64·7	138·6

That poverty is not necessarily a cause of infant mortality was clearly realised by William Cadogan, who, in his "Essay upon Nursing" (London, 1750), wrote: "In the lower class of Mankind, especially in the country, disease and mortality are not so frequent either among adults or their children. . . . The fact of superfluity confines them more within the limits of Nature. . . . The mother who has only a few rags to cover her child loosely and little more than her own breast to feed it, sees it healthy and strong . . . while the puny insect, the heir and hope of a rich family, lies languishing under a load of finery that overpowers his limbs, abhorring and rejecting the dainties he is crammed with, till he dies a victim to the mistaken care and tenderness of his fond Mother."

Density of Population and Overcrowding.—In considering the influence of overcrowding upon infant mortality we must distinguish between *density of population*, which means the *number of people per unit of land area*, and *overcrowding*, by which is to be understood *the number of people inhabiting a unit "room area."* It is clear that the two terms are not by any means synonymous, because of two districts having the same

density of population, the one possessing more many-storied houses will have a smaller degree of overcrowding than the other in which the number of such houses is fewer. William Farr, in 1843, in the Fifth Annual Report of the Registrar-General, pointed out that the general mortality is proportional to the sixth root of the density of population. Subsequently (in the Decennial Supplement) he modified his formula and substituted the eighth for the sixth root, as follows:—

$$\frac{m^1}{m} = \left(\frac{d^1}{d}\right)^{0\cdot11998} = \left(\frac{d^1}{d}\right)^{0\cdot12} \text{ approximately,}$$

where m^1 and m are the mortalities in two different places and d^1 and d are the respective densities in persons per square mile. Recent work, however, has shown that there is no relation between the density of the population and either the general or the infant mortality rates.

Relationship between Housing and Infant Mortality.—The following statistics show that overcrowding, as measured by the number of persons living in an occupied room, is associated with high infant mortality. It is true that in some rural districts housing conditions are deplorable, and yet infant mortality is lower than in urban districts, but the fact is that on the whole

TABLE LXIV.

Housing and Infant Mortality (Newsholme).

Rooms per tenement	Proportion per cent. of total population living in each class of tenement			Proportion per cent. of total population with more than two occupants per room.		
	1891	1901	1911	1891	1901	1911
	Aggregate of Urban Districts.					
1	2·9	2·0	1·7	1·6	1·0	0·7
2	9·4	7·4	6·5	4·4	3·1	2·5
3	11·5	10·3	12·7	3·5	2·6	3·0
4	22·4	21·2	23·1	2·8	2·2	2·2
	46·2	40·9	44	12·3	8·9	8·4
	Aggregate of Rural Districts.					
1	0·4	0·2	0·2	0·3	0·1	0·1
2	5·6	3·9	3·5	2·5	1·5	1·2
3	10·1	8·1	10·8	2·8	2·0	2·2
4	26·3	24·0	25·5	2·9	2·2	2·1
	42·4	36·2	40·0	8·5	5·8	5·6

TABLE LXV.—OVERCROWDING AND MORTALITY UNDER 1 AND BETWEEN 1 AND 5 YEARS.

		Number of rooms			
		1	2	3	4
Death-rate per 1,000 living	Under 1 year.. ..	210	164	129	103
	1-5 years	41	30	18	10
Relative mortality	Under 1 year ..	100	78	61	49
	1-5 years.. ..	100	73	44	24

DEATHS OF CHILDREN FROM VARIOUS CAUSES.

Cause of death	Number of rooms			
	1	2	3	4
Pneumonia	6	6	2	1
Infectious diseases	16	12	17	3
Diarrhœa and enteritis	32·8	25·8	14·9	16·8
Respiratory diseases	39·8	35·5	26·7	20·0

rural districts are better off in respect of overcrowding than are urban communities (see Table LXIV). Thus, in 1891, seven times as many lived in one-room tenements in urban as in rural districts (2·9 : 0·4). In 1901 the proportion was 10 : 1 (2 : 0·2), and in 1911, 8·5 : 1 (1·7 : 0·2). It can be shown that for twenty-nine metropolitan boroughs the correlation coefficient between overcrowding and infant mortality is $0{\cdot}69 \pm 0{\cdot}065$. (See p. 74.)

It has been alleged, by Pearson and others, that the association between bad housing and infant mortality is not one of cause and effect but the result of a common hereditary cause, viz., inferiority of stock, both physically and mentally, which determines the shifting of these people into inferior houses; but the fact that overcrowding is also associated with a high death-rate of children between 1 and 5 years of age entirely disposes of this contention (see Table LXV).

Further, the death-rates from pneumonia and the chief infectious diseases of childhood are proportional to the degree of overcrowding.

Dr. L. W. Darra Mair,[1] finds the infant mortality higher in back-to-back houses than in through houses. Miss Elderton

[1] "Report on the Relative Mortality in Through and Back-to-Back Houses," in Local Government Board Report, 1910.

[2] Ethel M. Elderton, "On the Relative Value of the Factors which influence Infant Welfare," *Annals of Eugenics*, vol. i, 1925, p. 139.

suggests that owing to the cheaper rental of the back-to-back houses the type of people inhabiting them is physically inferior to that occupying the "through houses." In other words, "man makes the environment and not the environment the man." But that Miss Elderton's suggested explanation is not the correct one is seen from the experience of Liverpool. In that city the infant mortality amongst inhabitants of certain slum areas was 300, but when the insanitary dwellings were demolished and *the displaced people* were accommodated in new and sanitary dwellings the infant mortality amongst them dropped to 167.

Hence it is fair to assume that with improved housing the infant mortality rate could still further be diminished in both urban and rural districts. It is to be noted, however, that in Eastbourne, where the density of population per house has increased from 6·1 in 1901 to 6·3 in 1921, infantile mortality has declined from 133 in 1898-1900 to 69 in 1923, so that the influence of other factors may overcome that of overcrowding.

The influence of housing and overcrowding is also shown by the following statistics (Children's Bureau of the U.S. Department of Labour (1915), Infant Mortality Series, No. 3) :—

TABLE LXVI.

Housing condition	Infant mortality
Clean, dry	105
,, damp	127
Moderately clean, dry	171
,, ,, damp	158
Dirty, dry	162
,, damp..	204
Water supply in house	118
,, ,, outside	198
Water closet	108
Yard privy	159
Number of others sleeping in same room with baby :—	
2 or less	67
3-5	98
Over 5	123
Baby sleeping in separate bed ..	56
,, not ,, ,, ,, ..	109
Ventilation of baby's room :—	
Good	28
Fair	92
Poor	169

Alcoholism in Relation to Poverty and Infant Mortality.— To modify a statement of Dryden's, "Intemperance is sure to poverty near allied." Which is the cause and which the effect is outside the province of our discussion in this place, but it is important to consider how far parental alcoholism is a factor in the causation of infant mortality. The following results of Dr. Sullivan show very clearly the progressively augmenting character of the influence of maternal alcoholism :—

TABLE LXVII.

	Number of mothers	Number of children	Number of children dead in two years	Percentage of dead children
Drunken mothers ..	21	125	69	55·2
Sober mothers ..	28	138	33	23·9

It has also been suggested that the cause of excessive infant mortality in mining areas such as Durham, where wages are high, as compared with the low infant mortality in such a place as Cambridgeshire, where wages are low, is due to the greater prevalence of drunkenness in the mining areas ; e.g., in 1911 there were about seven times as many convictions for drunkenness (in proportion to the population) in Durham as there were in Cambridgeshire (viz., 69·7 and 10·5 per 10,000 respectively). It must be remembered, however, that as convictions vary with the police administration such statistics are not quite reliable. Dr. Gibson, M.O.H. of North Riding combined districts, however, finds that the net or partial correlation coefficient between alcohol consumption per head and non-diarrhœal infant mortality (food consumption and winter temperature being kept constant), is as high as $0·90 \pm 0·03$. It is open to doubt whether the relationship is of such a high order as this, but everyday experience as well as common sense goes to show that the domestic neglect entailed by alcoholic indulgence (especially of the mother) must have some considerable influence upon infant mortality. There can be no question, for instance, that **overlaying** of infants is much more common on Saturday nights, when many mothers come home drunk, than on any other night.

The industrial employment of women is generally believed to be a factor influencing infant mortality, but statistics do not confirm this opinion, since whilst it is true that in certain districts such as Lancashire where the percentage of employed

women is high the infant mortality is also high, it is equally true that there are certain places where the correlationship is negative (see Tables LXVIII-LXX).

The reasons for this negative correlationship are probably that the unemployed women have larger families and less money to look after them than those who are employed.

American Statistics relating to the Influence of Maternal Employment upon Infant Mortality Rates.—Dr. Woodbury, of the U.S. Children's Bureau, gives the following figures based upon the study of 24,000 births :—

TABLE LXVIII.

Condition of mother's employment	Death-rate of infants			
	Neo-natal mortality	Early infant mortality	Total infant mortality	Premature births per cent. of total
Employed away from home ..	63·2	50·3	133·7	6·1
Employed at home	36·7	27·5	91 9	3·5
Not employed	43·1	35·6	93·3	5·2

Miss Elderton[1] finds that the partial coefficient of correlation between the mother's employment and the chances of the infant's survival (when the mother's age and health are constant) is only $0·10 \pm 0·05$, which is not significant.

The following are English statistics :—

TABLE LXIX.—(1)

Place	Percentage of employed women	Infant mortality
Wigan (1913)	12	180
Rochdale (1913)	28	106

TABLE LXX.—(2) DR. JESSE DUNCAN'S STATISTICS.

	Number	Infant mortality		
		1910	1909	1908
Mothers employed ..	731	153	179	190
Mothers unemployed	545	161	169	207

[1] Ethel M. Elderton : "On the Relative Values of Factors which influence Infant Mortality," *Annals of Eugenics*, vol. i, 1925, p. 23.

Illegitimacy.—In all countries the death-rate amongst illegitimate infants during all stages of infancy and from every cause is much higher than amongst legitimate ones. The causes of this are pre-maturity, wilful neglect, syphilis and lack of maternal breast-feeding. It has been found that the incidence of syphilis is eight times higher amongst illegitimate than the legitimate. In Moscow there is a great foundling hospital, containing 1,200 beds, and 98 per cent. of the admissions are of illegitimate infants. In 1914 it was found that the death-rate of those infants suckled by their mothers was 120 per 1,000 as compared with 305 per 1,000 amongst those suckled by wet-nurses. From these statistics we learn that, as far as possible, homes for unmarried mothers (of which there are at present 103, with accommodation for 1,305 mothers and 1,316 babies) ought to have accommodation for the mothers to remain with their babies throughout the greater part of infancy. There were 31,522 illegitimate babies born in 1923, and the mortality amongst them was 137 per 1,000 in the first year. It should have been possible to reduce their mortality to that of legitimate babies, viz., 68 per 1,000. (See Table XLVIII, p. 146.) This would have meant a saving of $(137 - 68) \times 31{\cdot}5 = 2{,}175$ infants.

TABLE LXXI.—INFANT MORTALITY PER 1,000 IN ENGLAND AND WALES BY LEGITIMACY.

Years	Legitimate	Illegitimate	Total
1906	127	261	132
1907	113	220	118
1908	116	233	120
1909	104	211	109
1910	102	195	105
1911	125	245	130
1912	91	181	95
1913	104	213	108
1914	100	207	105
1915	105	203	110
1916	87	183	91
1917	90	201	96
1918	91	163	97
1919	84	187	89

Total legitimate births in 1919 = 650,562.
Total illegitimate births in 1919 = 41,876.

It must be observed that the mortality of illegitimate infants is probably higher even than the figures given in this table, since a number of illegitimate infants are legitimized by the subsequent marriage of the parents.

Adoption.—It has been well said that the term illegitimate as applied to the child is a misnomer. "There may be illegitimate parents, but not illegitimate children." Dr. H. Dwight Chapin pleads for the adoption of illegitimate children—after the weaning age—on the ground that "being brought up in a precarious manner by the hard struggles of an unmarried mother, with the stigma of illegitimacy hanging over its head is not a happy outlook." Chapin believes that very few of these young unmarried mothers can really be considered bad. "They are rather ignorant and unsophisticated, and give for love what many better-placed women give for position and fortune. There is no connection between this class of women and prostitutes, who usually cannot have children if they would."[1]

In Norway efforts are made to establish the paternity of the child, and, as a result, 40 per cent. of the illegitimate children in that country receive support from their fathers, and until this principle is universally adopted, adoption of the child into a good family is probably the best solution of the problem in the majority of cases.

Sex-ratio as a Factor in Infant Mortality.—Statistics taken all over the world show that while more male than female babies are born every year, the male mortality is at all ages of infancy higher than that of females. The reason for this is not easy to ascertain, but the fact that the greatest difference in mortality between the sexes occurs during the neo-natal period suggests that there must be some ante- or intra-natal factor responsible for it. The greater size of the fœtal head is a possible explanation. If so, better ante-natal supervision (estimation of relative size of fœtus and maternal passages, preferably by X-ray examination) and improved obstetric technique should reduce the male mortality to that of female infants, viz., a reduction of 18 per 1,000 (= 78 − 60) with a saving of 6,970 male infants per year.

Climate and Infant Mortality.—In comparing infant mortality in different countries, the differences in climate must always be considered. New Zealand, for instance, has a climate favourable to low infant mortality, as it has also more favourable housing conditions.

Heat.—It has been shown by Liefman and Linderman that heat has a great influence on infant mortality. Poor housing

[1] Henry Dwight Chapin, "Heredity and Child Culture," London, 1923, p. 182.

conditions such as overcrowding, narrow streets and absence of thorough ventilation, which lead to high indoor temperature, undoubtedly increase the death-rate of infants. Dr. Gibson (*loc. cit.*) finds that the correlation coefficient between diarrhœal mortality and summer temperature is $+ 0{\cdot}87 \pm 0{\cdot}04$; and the coefficient between non-diarrhœal infant mortality and winter temperature is $- 0{\cdot}82 \pm 0{\cdot}05$.

Heredity and Infant Mortality.—The following table (LXXII) points to a relationship between longevity in the parents and the vitality of their children :—

TABLE LXXII.—(1) PERCENTAGE OF CHILDREN WHO DIED IN THE FIRST FIVE YEARS OF LIFE ACCORDING TO AGE AT DEATH OF EITHER PARENT (3,855 CHILDREN).

	Age of parent at death					
	All ages	to 38	39-53	54-68	68-83	84 and over
Per cent. for mothers ..	28	52	38	30	20	10
Per cent. for fathers ..	26	40	34	27	22	17

(2) SAME PERCENTAGES FOR ROYAL FAMILIES (3,200 CHILDREN).

	Age of father at death								
	All ages	16-25	-35	-45	-55	-65	-75	-85	86 and over
Percentage of children dead before 5 years old	28	52	32	31	31	28	26	24	3

See further, Chapter VII, p. 246.

TABLE LXXIII.—MORTALITY OF CHILDREN FOR EACH OF THE FIRST FIVE YEARS OF LIFE, PER 1,000 AT EACH AGE.

Period	0-1	1-2	2-3	3-4	4-5
1871-1875	154	59	28	19	14
1876-1880	145	58	27	17	13
1881-1885	139	53	23	15	12
1886-1890	145	53	22	14	10
1891-1895	151	52	21	14	10
1896-1900 . ..	156	49	19	13	9
1901-1905	138	41	16	11	8
1906-1910	117	35	14	9	7
1911-1915	110	35	14	9	7
1921	85	19	9	6	4

This table shows that the decline in mortality of children after the age of 1 year began earlier than that of infant mortality.

TABLE LXXIV.—SPECIFIC DEATH-RATES FOR AGE-GROUPS UNDER 5 YEARS, BETWEEN 1909 AND 1923. NEW YORK CITY. (From data by Josephine Baker.)

Year	Mortality rate per 1,000 of the given age							
	0-1		1-2		2-4		0-5	
1909	137	Average 116·4	50	Average 41·2	13	Average 11·8	49	Average 41·8
1910	126		45		13		48	
1911	112		39		12		43	
1912	105		36		10		30	
1913	102		36		11		39	
1914	95	93·4	32	33·2	9	10·6	37	36·6
1915	98		34		9		38	
1916	93		33		12		37	
1917	89		28		9		33	
1918	92		39		14		38	
1919	82	75·8	22	20·4	9	7·4	28	25·6
1920	85		28		9		31	
1921	71		16		7		24	
1922	75		23		7		25	
1923	66		13		5		20	
Reduction per cent. between 1909 and 1923	51·8		74		61·5		59·2	

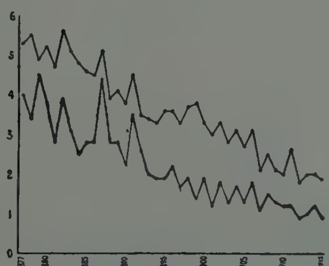

FIG. 35.—Death-rate per 1,000 per annum, during the years 1877-1914, in Huddersfield. Upper line represents death-rate at ages 0—1 year; lower line represents death-rate at ages 1-5 years. (S. G. Moore, *Lancet*, March 14, 1916.)

The Relation between Infant Mortality and Mortality at Higher Ages.—If we look at the diagram (fig. 35), we see that the curve of infant mortality runs a practically parallel course to that of the total mortality of children between 1 and 5 years. Moreover, examination of statistics in different parts of the country as well as in different periods of time, shows that in those localities (or years) in which infant mortality is high, the mortality

at subsequent ages of childhood and youth is also high, and vice versa. (Tables LXXIII and LXXIV.)

Birth-rate and Infant Mortality in Relation to Birth Control.—One of the principal arguments of those who advocate the widespread dissemination of birth-control information amongst the public is the alleged causal relationship between high birth-rate

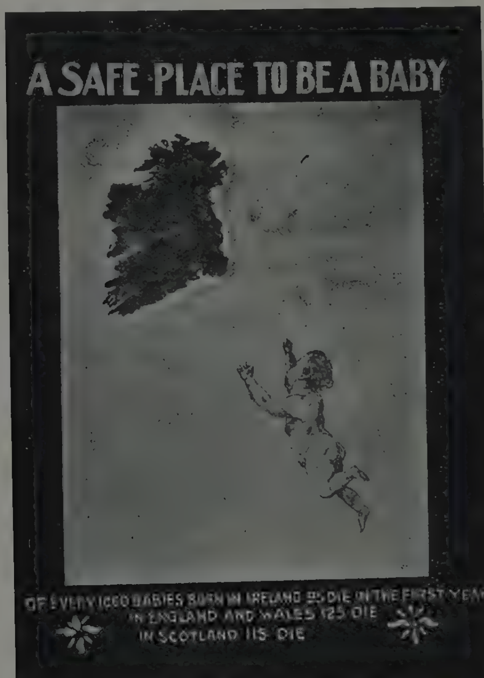

FIG. 36.—The mortality figures in the illustration are those for the years 1903—1913.

and high infant mortality, and the difference between an infant mortality of 80 in South Africa and one of 40 in New Zealand has recently been ascribed as being partly due to a difference between a birth-rate of 29·8 in South Africa and one of 25·5 in New Zealand. But whilst it is true that a high birth-rate is often associated with a high infant mortality rate, this is not

invariably the case. In England and Wales the birth-rate has been falling since 1841, but infant mortality only began to fall steadily since 1900. In Ireland, as well as in other parts of the world, the infant mortality is low in spite of the high birth-rate (fig. 36). In New Zealand the birth-rate has remained practically stationary, but the infant mortality rate has been declining since 1907. In Japan the birth-rate has recently been steadily declining to 32·2 per 1,000 in 1918, but the infant mortality rate has increased from 160 in 1915 to 189 in 1918. Dr. Willoughby, M.O.H. for Eastbourne, finds that between the quinquennia 1896-1900 and 1919-1923 the birth-rate in Eastbourne has declined by 6·1 per 1,000, whilst the infant mortality rate has fallen by no less than 80 per 1,000, a totally disproportionate fall.[1] Moreover, he finds that while the birth-rate has declined mostly amongst the well-to-do, who could in any case afford to look after more children, the reduction in infant mortality in Eastbourne has been chiefly amongst the poor and ignorant, who still have large families, but amongst whom infant welfare work has been most energetically undertaken. It is true that the greater the number of children amongst the poorer classes the less is their chance of survival, because of the inability of the mother to devote her attention to them, but it is equally true that the number of births in a family is often determined by the number of children that have died. Hence it is quite conceivable that it is not the birth-rate which determines the size of the infant mortality, but that the latter determines the former.

Brownlee[2] finds also that there is no evidence that large families are more unhealthy than small ones.

My own personal experience is that the infant mortality amongst large families is not necessarily higher than amongst small families of the same social status. It is quite a common experience to meet mothers of the industrial class who have given birth to ten or more children without losing one—during infancy or early childhood. Moreover, it is equally common to find that not only does such frequent child-bearing not undermine the mother's health—as is frequently alleged by the advocates of indiscriminate birth-control propaganda—but that they actually feel better and stronger with each pregnancy.

Economic Argument regarding Birth Control.—As regards

[1] *Journ. State Med.*, 1925, xxxiii.
[2] *Lancet*, November 1, 1924, p. 925.

the argument that an uncontrolled birth-rate would soon increase the size of the population of the world to such an extent that there would not be enough standing room or food for all, it is to be said that those who put forward such a contention either ignore or are ignorant of two essential facts. They erroneously assume that the growth of a population increases in geometrical progression. Were that the case it would indeed not take many generations before the world would become uncomfortably over-

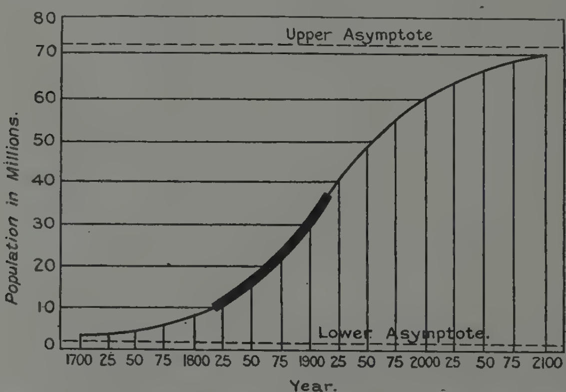

FIG. 37.—The population growth of England and Wales (after Raymond Pearl, "Studies in Human Biology").

The equation of the curve is :—

$$y = 2{\cdot}373 + \frac{70{\cdot}670}{1 + 9{\cdot}179e^{-0{\cdot}0195x}}$$

The thick portion of the curve represents the part drawn from the decennial census figures for the years 1801-1911. The values calculated from the equation are found to agree closely with the recorded census figures. The point of inflection, i.e., the point at which the rate of increase of population was greatest, was in the year 1916. The upper asymptotic population is approximately 73 millions, and in the year 2100 the population will reach the size of about 71 millions.

crowded. But it has been shown by Raymond Pearl and Udny Yule, independently, that population growth does not take place at an ever-increasing rate, but that, on the contrary, population increases at a rate which keeps on diminishing, tending towards some limit. Put in mathematical language, the curve of population has an asymptote parallel to the axis of time (fig. 37). According to Brownlee, if the birth-rate continues at the average level for 1921-23, and the death-rate at that of 1911, the population

in Britain will attain a maximum of 49,000,000 in 1971 (present population about 43,000,000) and then decline. Pearl estimates the population in England and Wales in 1971 as about 55,000,000, and the maximum which will be reached in the year 2100 as about 71,000,000.

Further than that, a glance at a map will show that not only is the world as a whole under-populated, but that even in temperate zones there are large regions in which vastly greater populations could be accommodated and maintained, even with the present possibilities of agriculture. Nobody, of course, can foretell what the future has in store in the way of agricultural inventions and organizations; it is quite likely that the tropical as well as the arctic regions might, with the progress of research, as yet become utilizable for cultivation purposes.

Eugenic Argument.—The last argument in favour of birth control is based upon the contention that with the present differential birth-rates amongst the poorer and richer classes there is a preponderance of births of children of inferior stock. This would be a very cogent argument in favour of widespread birth-control propaganda amongst the class of women who attend the infant welfare centres. But in our present state of knowledge it is not at all easy to decide which is the inferior and which the superior stock. The only certain inferiority is transmissible disease or insanity (including mental deficiency), but the birth of these defectives, which is by no means the sole prerogative of the poorer classes, ought to be prevented by other means (see Chapter VII, p. 231) rather than by widespread and indiscriminate teaching of contraceptive methods.

Number of Children per Family necessary to keep the Population Stationary.—Let us imagine that every male and female child that is born survives long enough to reach the marriage age; that all of them marry; that all such marriages are fertile; and that all conceptions result in the birth of living children, who go through the same cycle. Then, in order to keep the population stationary, it is obvious that every married couple must have two children to replace them. But since out of every 100 individuals born alive only seventy-three reach the marriage age, and only 85 per cent. of the survivors actually marry, also, only 93 per cent. of those that marry *can* become parents, and finally, only about 80 per cent. of the conceptions result in the birth of living children, it is obvious that the minimum number living children per couple, to maintain the population stationary, is $\frac{2 \times 100 \times 100 \times 100}{73 \times 85 \times 93} = 3{\cdot}5$

and the minimum number of conceptions or pregnancies required for the purpose is $3{\cdot}5 \times \frac{100}{80} = 4{\cdot}4$. In other words the minimum number of pregnancies per marriage should be between 4 and 5.

Race and Infant Mortality.—From Tables XXXIX and XL given on pp. 138-139, we learn that in a comparison of infant mortalities in different places, as well as at the same place at different times, the racial make-up of the places at the times in question must be taken into account. Thus, the immigration of Jews into New York has accelerated the rate of decline of infant mortality in that city quite apart from the improvement in health conditions or infant welfare activities. If Jewish infants are excluded from the statistics for 1915, the infant mortality rate for that year rises from 98 to 105.

We are now left with the other question that we set ourselves to solve, viz., **What is the limit to which we may hope to reduce the infant mortality rate?** The answer is certainly less than 40 per 1,000, since in certain areas of considerable size, where the infant mortality has not yet reached the lowest limit, it is as low as 40 per 1,000. For example, in Roscommon and Cavan, in Ireland, with populations of over 90,000, the infant mortality rates in 1914 were 38 and 40 respectively. In New Zealand it is about 40 per 1,000. I believe that in the present state of our knowledge it is impossible to expect a greater reduction than to about 20-25 per 1,000, since that figure represents approximately the number of infant deaths due to ante-natal causes the nature of which we do not as yet understand.

The Expectation of Life.—By the "expectation of life" is meant the number of years that a person of any given age will, on the average, survive. Pearl suggests that "mean after life-time" is a better term for this. Statistically it is designated by the symbol $\mathring{e}x$. Glover[1] gives the following table for the expectation of life at various ages of infancy and childhood (see Table LXXV).

This table shows that the expectation of life increases from birth to about the third year of life, when it reaches a maximum; after that it gradually decreases. It also shows that the expectation of life at 11 years of age is approximately the same as at birth. It further illustrates the fact that the specific death-rate

[1] J. W. Glover, United States Life Tables, 1890, 1901, 1910, and 1901-10, Washington (Bureau of the Census), 1921. Quoted by Pearl, "Medical Biometry and Statistics," 1923, p.180.

is highest at the extremes of life, and is a minimum at the age interval, 11-12.

TABLE LXXV.

Age interval	Expectation of life in years at beginning of the age interval	Specific death-rate
0- 1 months ..	51·49 years	43·77 per month
1- 2 ,, ..	53·76 ,,	11·83 ,,
2- 3 ,, ..	54·32 ,,	9·98 ,,
3- 4 ,, ..	54·78 ,,	8·57 ,,
4- 5 ,, ..	55·17 ,,	7·60 ,,
5- 6 ,, ..	55·51 ,,	6·90 ,,
6- 7 ,, ..	55·81 ,,	6·33 ,,
7- 8 ,, ..	56·08 ,,	5·87 ,,
8- 9 ,, ..	56·33 ,,	5·45 ,,
9-10 ,, ..	56·56 ,,	5·08 ,,
10-11 ,, ..	56·76 ,,	4·72 ,,
11-12 ,, ..	56·95 ,,	4·38 ,,
0- 1 years ..	51·49 years	114·62 per year
1- 2 ,, ..	57·11 ,,	27·62 ,,
2- 3 ,, ..	57·72 ,, (maximum)	12·34 ,,
3- 4 ,, ..	57·44 ,,	7·83 ,,
4- 5 ,, ..	56·89 ,,	5·65 ,,
9- 10 ,, ..	53·02 ,,	2·47 ,,
10- 11 ,, ..	52·15 ,,	2·27 ,,
11- 12 ,, ..	51·26 ,,	2·19 ,, (minimum)
12- 13 ,, ..	50·37 ,,	2·22 ,,
13- 14 ,, ..	49·49 ,,	2·36 ,,
14- 15 ,, ..	48·60 ,,	2·57 ,,
19- 20 ,, ..	44·34 ,,	4·28 ,,
24- 25 ,, ..	40·38 ,,	5·42 ,,
29- 30 ,, ..	36·48 ,,	6·28 ,,
39- 40 ,, ..	28·94 ,,	9·11 ,,
49- 50 ,, ..	21·69 ,,	13·77 ,,
59- 60 ,, ..	15·03 ,,	26·73 ,,
69- 70 ,, ..	9·58 ,,	35·14 ,,
79- 80 ,, ..	5·56 ,,	119·10 ,,
89- 90 ,, ..	3·20 ,,	254·31 ,,
99-100 ,, ..	1·95 ,,	385·46 ,,

Increased Expectation of Life at Time of Birth.—Rollo H. Britten (quoted by Josephine Baker) has shown that the expectation of life at birth for the white and coloured population of the Registration States of America has increased between 1900 and 1920, as follows :—

TABLE LXXVI.

Period	White		Coloured	
	Male	Female	Male	Female
1900-02	48·2	51·1	32·5	35·0
1909-11	50·2	53·6	34·1	37·7
1919-20	54·1	56·4	40·5	42·3

Expectation of Life at Different Ages of Infancy.—The following table is given by Whipple:—

TABLE LXXVII.

Age interval, months	Average length of life remaining to each one alive at beginning of age interval	
	Males	Females
0 – 1	49·86	53·24
1 +	52·35	55·28
2 +	52·96	55·78
3 +	53·46	56·20
4 +	53·88	56·56
5 +	54·24	56·87
6 +	54·56	57·15
7 +	54·85	57·41
8 +	55·11	57·64
9 +	55·35	57·85
10 +	55·57	58·05
11 +	55·76	58·22

TABLE LXXVIII.—FALL IN INFANT MORTALITY IN CERTAIN TOWNS AND COUNTIES GROUPED ACCORDING TO DATE OF COMMENCEMENT OF INFANT WELFARE WORK (JAMES WHEATLEY[1]).

Group and date when health visiting commenced	Decrease in five-year periods stated as percentage of period immediately preceding					
	1895-1900	1901-1905	1906-1910	1911-1915	1916-1920	1921-1922
(I) 1900-1905.. (13 towns)	+ 4·4	– 13·9	– 14·2	– 6·8	– 16·9	– 12·6
(II) 1906-1910.. (9 towns)	0·0	– 13·3	– 16·0	– 6·3	– 13·6	– 12·7
(III) 1912-1913.. (6 towns)	– 1·8	– 15·0	– 19·0	– 6·1	– 12·9	– 17·0
(IV) 1913-1916.. (22 counties)	...	– 12·7	– 10·3	– 9·5	– 15·8	– 11·25
(V) 1917-1920..	...	– 15·22	– 17·9	– 8·9	– 14·1	– 12·7

This table does not reveal any difference in time or rate of fall of mortality between the places where child welfare activities began early or late.

The Relation of Infant Welfare Clinics and Ante-natal Centres to Infant Mortality.—That the decline in infant mortality cannot be entirely due to campaign work in connection with infant welfare is seen from the fact that the fall began some time before the passing of the various Acts, such as the Midwives Act, Notification of Births Act, &c. (See Table XLII on p. 170.)

[1] Paper read before British Medical Association in 1923.

Moreover, Table LXXVIII shows that there has been very little difference in the time of commencement or extent of decline in infant mortality between those places in which infant welfare schemes began early or late in the century. On the other hand, it has been clearly shown that in the same area the infant mortality is much higher in those districts where there is little or no infant welfare work done than in those where the campaign is carried out with vigour. Hence we must conclude that, although the various clinics and centres have not done all that is being claimed for them by their enthusiastic partisans, they have accomplished a good deal more than their detractors will admit. In view of the fact that infant mortality began to fall some thirty years after the passing of the Education Act of 1870, it is possible that that Act may have had its good effect upon that generation of parents who were school children when that Act was passed.

Further, the fact that the decline in infant mortality commenced about 1900, i.e., about ten years before maternity and child welfare work was started, shows that, to some extent at any rate, the one cannot be due to the other. Possibly a great deal of the decline is due to general economic improvement of the people, improvement in milk supplies, increased national sobriety, the use of tarred roads and decrease of horse-traffic, with the concomitant decrease in the number of flies that thrive on fæcal excreta, as well as better knowledge of mothercraft on the part of the mother.

The Value of Infant Welfare and Ante-natal Clinics.—In estimating the value of these clinics, account must be taken of the ages and general health of the babies visiting these centres. An example will make this clear. In New York, in 1915, 108 special nurses visited the homes of some 16,000 infants for fifty-four days between July and September. The total number of deaths amongst these babies was 187 during the fifty-four days. This gives a mortality per year of:—

$$\frac{187}{16} \times \frac{365}{54} = 78 \text{ per } 1{,}000.$$

The mortality of unsupervised infants during the same *summer* period was 140 per 1,000 per year, and therefore there was apparently a saving of 62 per 1,000 infants effected by these home visits, viz. (140 − 78). But this is a gross exaggeration of the beneficial results of supervision, because, whilst the unsupervised babies were of all ages between birth and 1 year, those visited were between $5\frac{1}{2}$ and 7 months old, amongst whom the

mortality is 34 per 1,000 less than the total mortality for the infants of all ages. Hence the real mortality of the unsupervised infants of the same age-group was only 106 per 1,000 per year (i.e., 140 − 34) during the summer period, so that the saving effected by supervision, instead of being 62 per 1,000 was, in any case, not more than 28 per 1,000. Moreover, 87 per cent. of the supervised infants were breast-fed before enrolment, as against only 80 per cent. in the whole of the city, so that the average of the babies visited were in better health than those unsupervised. It is therefore probable that the net saving resulting from home visiting was about 20 per 1,000 per year, instead of the apparent 62 per 1,000.

By the same method of calculation it can be shown that whilst the mortality amongst the babies that were brought to the clinics was 65 per 1,000 per year during the summer months, as compared with 140 per 1,000 per year during the same period, showing an apparent saving of 78 per 1,000 effected by these clinics, yet when allowance is made for the difference in ages between the two classes of infants, the net saving was 32 per 1,000.

It is obvious that as the babies that are brought to the infant welfare centres can hardly be less than one month old, the mortality amongst them (as seen from Table XLIII, on p. 143) must in any case be no more than 60 per cent. of the total infant mortality for the babies (from birth to one year) not attending these centres. Even if they were brought to the centre after the first week, the mortality amongst the "centre" babies cannot—quite apart from any benefit derived from the attendance at the centre—be more than about 75 per cent. of the total infant mortality.

It must, however, be remembered that the total saving of infant lives resulting from such supervision cannot be measured by the above figures alone, since the instruction given to the mothers during the period of supervision must enable them to care for their babies better after the supervision has ceased. Moreover, the reduction of morbidity amongst the supervised infants must result in their improved health and physical fitness.

Indeed, a comparison of the infant mortality rates in places where intensive infant hygiene work has been carried out with those in places where no such work is undertaken, shows a reduction of infant mortality at all stages of infancy, as seen from Table LXXIX. Drs. Brend and Findlay doubt the value of these clinics because, as they point out, the variations in infant mortality in places where milk depots and infant clinics have been instituted (such as Bradford) have been practically the same as for the whole of England and Wales, but the comparison is not a fair one, because

we do not know what proportion of the infants in such cities attend these welfare centres or their ages. It may possibly be only a very small proportion, and has therefore very little effect upon the death-rate. It would be fairer to compare the mortalities in the same city of the infants who do, and those of the same age and same social standard who do not, attend the welfare centres. The American statistics do make such a comparison.

It will be seen that numerous factors enter into infant welfare work, and one or more favourable factors may compensate for other unfavourable ones, and vice versa.

TABLE LXXIX.—INFANT MORTALITY FOR ENGLAND AND WALES AS A WHOLE AND FOR BRADFORD SEPARATELY.

Year	Deaths under one year per 1,000 births	
	England and Wales	Bradford
1881-1890	142	165
1891-1900	153·5	170·5
1901	151	168
1902	133	139
1903	132	148
1904	145	167
1905	128	144
1906	132	152
1907	118	124
1908	120	143
1909	109	116
1910	105	127
1911	130	140
1912	95	99
1913	108	128
1914	105	122
1915	110	123

Is Infant Welfare Work worth while ?—It has been urged by some that the reduction of infant mortality brought about by the various efforts is not as great a blessing as it is supposed, since the babies that are saved during the first year of life are so feeble, coming as they do from feeble stock, such as tuberculous and other sick parents, that they die during the subsequent years of early childhood (1-5 years). Moreover, it is contended that even if any of them manage to reach adult age, they become the parents of weaklings like themselves, thus assuring the perpetuation of the unfit. Infant mortality, they say, is selective, killing off the unfit, and should not be interfered with. But each of these arguments is untenable. The fact that there is a co-variation

between the mortality of early infancy and that of later infancy, as well as between total infant mortality and the mortality of children of the ages 1-5, so that in places where one is high the others are high and vice versa (see Tables LXXIII and LXXIV, and fig. 35), disposes of the selective mortality argument, As for the second argument, whilst the saying *Mens sana in corpore sano* is an excellent slogan to be used in a campaign for the improvement of physical fitness, it must be remembered that some of the best brains in the world, e.g., those of R. L. Stevenson, Darwin, Nelson and others, dwelt in the frailest of bodies.

Measures for the Preservation of Infants' Lives and Health.—An intensive campaign in child welfare should include the following:—

(1) Provision of free continuation classes in infant welfare which all girls should be encouraged to attend. The instruction to be by means of diagrams, lantern slides, moving pictures, &c. (fig. 38).

Fig. 38.—Cinematograph film to teach infant welfare work.

All these are taken from a maternity film shown at girls' schools by the Public Health Department of Bermondsey. The whole film shows a mother attending an ante-natal clinic where she is examined by the doctor. Then suitable maternity garments are shown. Then the care of the baby with regard to its bath, its dressing and its feeding, and its being put to bed.

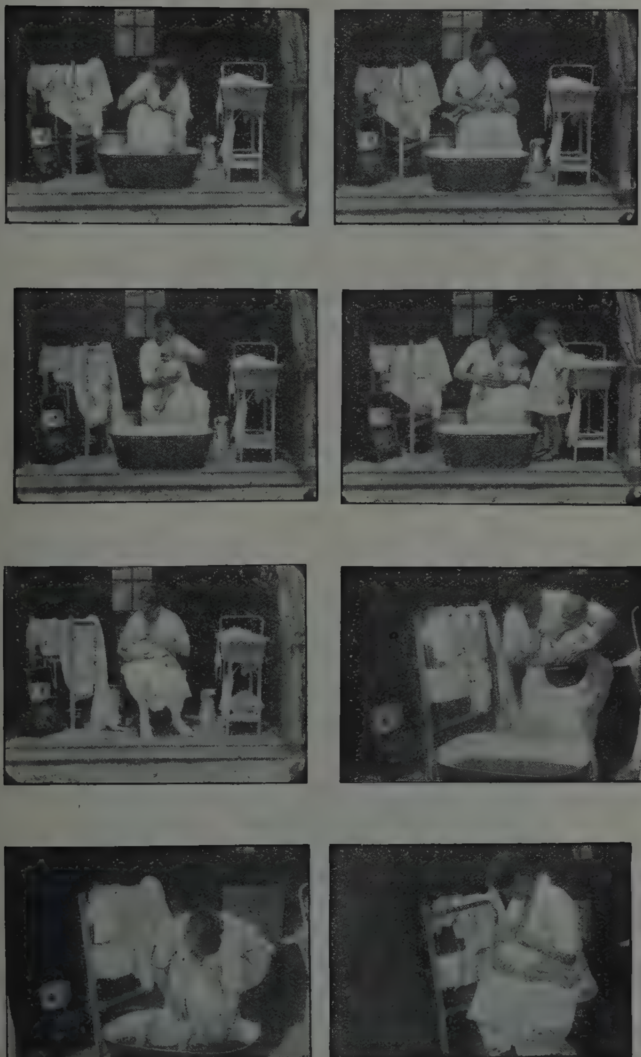

FIG. 38—*continued.*

FIG. 39.—Teaching mothercraft in Russia.

FIG. 40.—Teaching mothercraft in Russia.

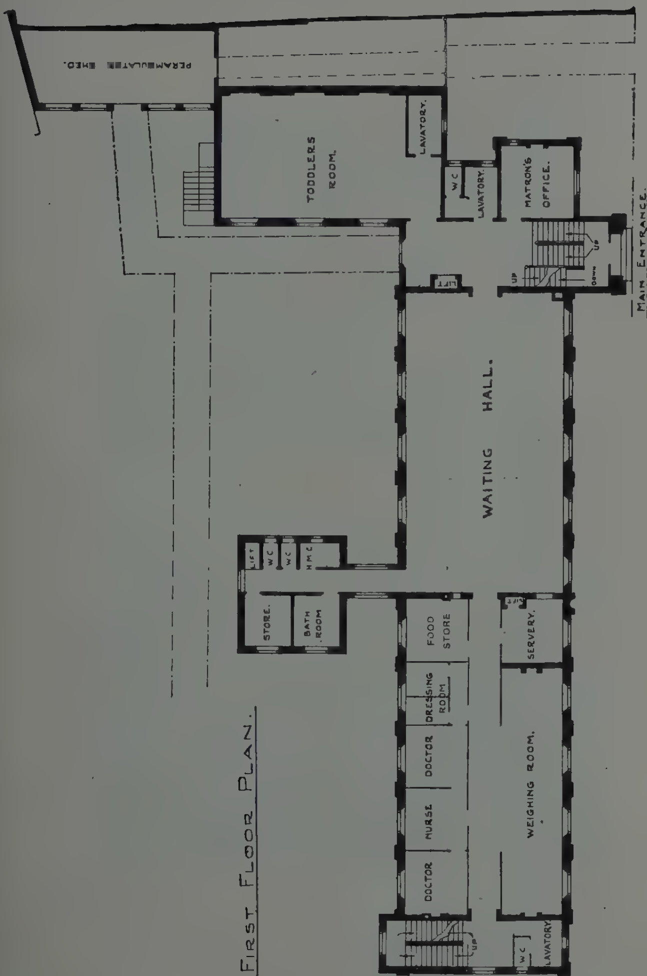

FIG. 41.—Plan of Birmingham Infant Welfare Centre.

(2) Child hygiene and mothercraft to be a subject of tuition in all schools, especially in secondary schools (figs. 39 and 40).

(3) Ante-natal and post-natal child physiology and hygiene in their theoretical and practical aspects ought to be subjects of instruction in the medical schools.

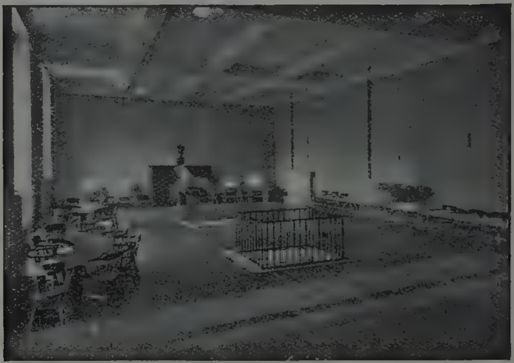

Fig. 42.—Birmingham Infant Welfare Centre. Toddlers' room.

Fig. 43.—Birmingham Infant Welfare Centre. The observation ward.

(4) Provision of good, commodious and hygienic houses at reasonable cost or rental.

(5) With the supply of these needs the necessity for the various infant welfare centres will diminish ; but until that desir-

able state of affairs arrives I should like to see these centres presided over by full-time medical officers who have made a speciality of the study of infant physiology and hygiene and who have a genuine love for children. Figs. 41 to 44 show the plan and rooms of a model infant welfare centre. In 1925 there were altogether 2,195 infant welfare centres in England and Wales,

FIG. 44.—Birmingham Infant Welfare Centre. The sun balcony.

as follows: Under county councils, 561; under local sanitary authorities, 861.

It is never safe to prophesy, but I think one may predict with considerable confidence that before many years have passed the whole of later infant mortality (i.e., between 1 and 12 months) will be practically wiped out, and the neo-natal mortality will be greatly reduced, so that the total infant mortality will, as the

result merely of the application of knowledge we already possess, be lowered to about 30 per 1,000.

The Plunket system in New Zealand, named after Lord Plunket, the Governor in 1907, and Lady Plunket,[1] and with which the name of Sir F. Truby King is to be honourably associated, consists of:—

(*a*) Home visiting by "Plunket" nurses, who give instruction in all branches of the art of motherhood.

(*b*) Complete system of birth registration.

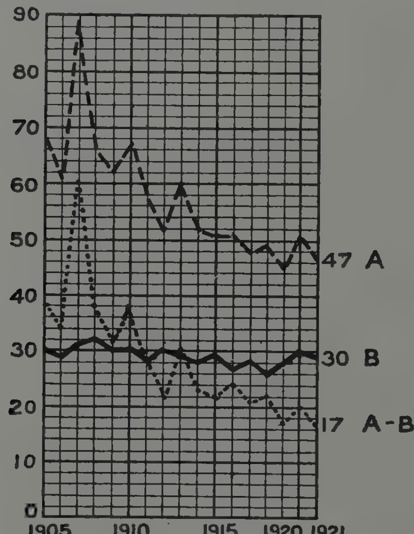

FIG. 45.—I. New Zealand. (For meaning of letters see fig. 46.)

(*c*) Close supervision of midwives, maternity hospitals, infant institutions and nurses. This system claims to have succeeded in reducing the infant mortality from about 70 in 1904-08 to about 40 in 1922. It must, however, be stated that whilst the system was introduced about 1907, infant mortality in New Zealand had previously been gradually declining from 107 in 1871-75 to 71 in 1904-08, a fact entirely due to other factors, such as housing and sanitation. It cannot be denied, however, that the educational value of the system was responsible for the

[1] The official name of the society which adopts that system is the Royal New Zealand Society for the Health of Women and Children. A full account of its work is given in *The Times* of June 5, 1926.

decline in infant mortality between 1908 and 1922 to a far larger extent than its detractors will admit.

Even in New Zealand the decline in neo-natal mortality has been very slight indeed, but during the last few years special

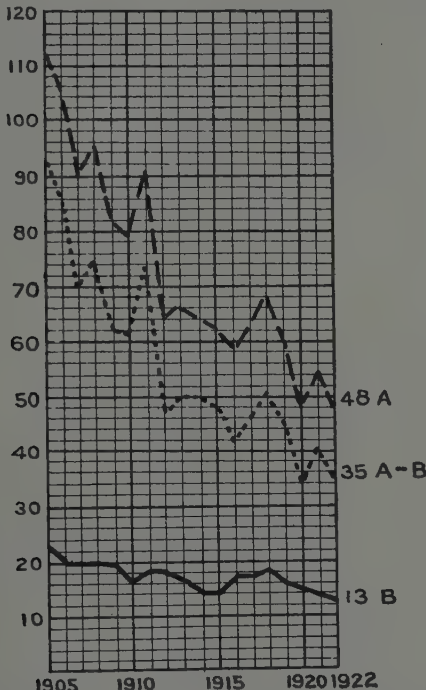

FIG. 46.—II. Amsterdam.

Two charts to compare the fluctuation of infant mortality in New Zealand and Amsterdam during the last 15 years.

A = total mortality-rate under one year
B = mortality-rate in the first month of life (neo-natal rate).
A-B = mortality-rate of infants from 2-12 months.

attention has been given to ante-natal work and neo-natal visiting. The results of such supervision will be watched with interest. Amsterdam seems to be the only place where the neo-natal mortality fell from 23 per 1,000 in 1905 to 13 per 1,000 in 1922 (fig. 46).

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CHAPTER VI

MATERNAL MORTALITY

"It falls out often, that in such dangerous travails, one, three, or two lose their lives, besides the loss a good Husband hath in his Wife and poor children in their Mother, &c. And in case such parties may escape with their lives in hard travails, yet are they so pulled and torn, that they are made unfit for any work which otherwise might have better been preferred, if honestly and faithfully they had been dealt withal. This I speak not as an invective against others; but, every one look to it, what they are intrusted withal, and make a conscience in their waies, remembering also that they must be accounted unto God for it, then they need not to be put in mind of it by my words."—FELIX WÜRTH (sixteenth century).

CLOSELY associated with the subject of infant mortality is that of maternal mortality, by which is understood the death of women from causes associated with pregnancy and labour. The following table of **Causes of Maternal Mortality** is given by Sir George Newman in his Annual Report to the Minister of Health for 1923 :—

TABLE LXXX.

Causes of Maternal Mortality (England and Wales, 1923).

Cause of death	All ages	15—	20—	25—	30—	35—	40—	45—
Abortion	100	2	7	17	38	22	14	—
Ectopic gestation ..	97	1	10	18	30	26	9	3
Other accidents of pregnancy	152	3	20	21	30	37	36	5
Puerperal hæmorrhage	432	7	31	76	121	116	73	8
Other accidents of childbirth	304	2	44	63	61	82	46	6
Puerperal sepsis ..	985	27	167	238	256	186	99	12
Phlegmasia alba dolens	61	—	7	13	16	15	10	—
Puerperal embolism and sudden death	216	—	28	42	69	47	25	5
Puerperal nephritis and uræmia	120	3	19	25	28	32	9	4
Puerperal albuminuria	40	—	6	6	9	15	4	—
Puerperal convulsions	358	18	77	97	80	59	25	2
Other causes	27	—	2	5	9	8	2	1
Total	2,892	63	418	621	747	645	352	46

The Measurement of Maternal Mortality.—As maternal mortality rate is meant to show the danger associated with pregnancy and labour, the most ideal method of measuring it would be by the number of maternal deaths per 1,000 pregnancies (i.e., labours at full term, together with abortions and miscarriages, as well as other deaths unaccompanied by the expulsion of a fœtus). This ideal method, however, is, in the absence of registration of pregnancy and abortion, unattainable. The denominator used is therefore the number of live births, and we hence define **maternal mortality** rate as **the number of maternal deaths per 1,000 live births.** There is a slight error associated with this method of measurement, since no account is taken of multiple births, but the error is so slight as to be negligible. It must, however, be recognized that as medical practitioners are often averse to issue a certificate giving parturition as a cause of death, the figures of maternal mortality rates are somewhat of an under-estimate.

Trend of Maternal Mortality.—The following figures (Table LXXXI) give the trend of the total maternal mortality between 1861 and 1923:—

TABLE LXXXI.

Period	Maternal mortality	
	Per 1,000 live births	Ratio based on rate per 1,000 live births
1861-1870	4·73	100
1871-1880	4·73	100
1881-1890	4·78	101
1891-1900	5·11	108
1901-1905	4·27	90
1906-1910	3·74	79
1911-1915	3·81	81
1916-1920	3·88	82
1921	3·71	80
1922	3·58	76
1923	3·60	76

The actual or *net* number of maternal deaths has diminished between 1900 and 1923 from 4,455 to 2,732, because the birth-rate between those dates has decreased from 28·7 to 19·7 per 1,000 persons, but the maternal mortality *rate* has shown very little decline. From Tables LXXX and LXXXII we see that the highest mortality from any single cause is—as has always been the case—from puerperal sepsis; it, together with phlegmasia alba

dolens, having been responsible in 1923 for 985 + 61 = 1,046 out of the total 2,892 cases, constituting a proportion of 36 per cent., although as a percentage of total maternal mortality it has decreased from 45 per cent. in 1900 to 36 per cent. in 1923.

TABLE LXXXII.—MATERNAL MORTALITY IN ENGLAND AND WALES BETWEEN 1900 AND 1923, DIVIDED INTO MORTALITY FROM SEPSIS AND THAT FROM OTHER CAUSES.

Year	Maternal Mortality		
	(a) Sepsis	(b) Other causes	Total
1900	2·18	2·63	4·81
1901	2·49	2·19	4·73
1902	2·13	2·34	4·47
1903	1·76	2·31	4 07
1904	1·75	2·27	4·02
1905	1·87	2 33	4·20
1906	1·75	2·27	4·02
1907	1·60	2·23	3·83
1908	1·48	2·09	3·57
1909	1·56	2·14	3·70
1910	1·42	2·14	3·56
1911	1·52	2·15	3·67
1912	1·47	2·31	3·78
1913	1·34	2·37	3·71
1914	1·63	2·32	3·95
1915	1·56	2·38	3·94
1916	1·47	2·40	3·87
1917	1·39	2·27	3·66
1918	1·35	2 20	3·55
1919	1·76	2·36	4·12
1920	1·87	2·25	4·12
1921	1·46	2·25	3·71
1922	1·46	2·12	3·58
1923	1·38	2·22	3·60
1924	1·48	2·22	3 70
1925	1·62	2·24	3·86

TABLE LXXXIII. — COMPARISON OF MATERNAL MORTALITY RATES IN ENGLAND AND WALES AND THE UNITED STATES OF AMERICA (PER 1,000 LIVE BIRTHS).

	1901-05	1905-10	1915	1916	1917	1918	1919	1920
England and Wales	4·3	3·7	3·9	3·9	3·7	3·6	4·1	4·1
U.S. Registration Area	—	—	6·1	6·2	6·6	9·2	7·4	8·0

Puerperal Septicæmia.—Reference to puerperal septicæmia is found in the writings of Susruta (second century B.C.) and

Hippocrates (400 B.C.). J. D. Rolleston[1] quotes the following epitaph by Leonidas (vii, 463) illustrating the high puerperal mortality (probably due to sepsis) at that time: "Here lie Timoclea, Philo, Aristo, and Timetho, daughters of Aristodicus, all slain by birth pangs." According to Mauriceau, in an epidemic of puerperal fever in 1660 in the Hôtel Dieu, in Paris, 66 per cent. of the women delivered there died of this disease. The Hippocratic view, which prevailed up to the seventeenth century, was that the disease was caused by the suppression of lochia—a confusion, as we now know, between effect and cause. From that time till the middle of the nineteenth century it was believed that suppression of mammary secretion was responsible for the disease—a symptom having again been mistaken for the cause. In 1843, Oliver Wendell Holmes not only showed that the disease was caused by the transference of infection from decomposing material to the mother, but also formulated rules for its prevention. His teaching, however, was not accepted. In 1847, Semmelweiss, who was assistant in the maternity clinic in Vienna, noticed that in that department of the clinic in which labour and puerperium were attended by medical students the maternal mortality was 11·4 per cent., whilst of the women attended by midwives only 2·7 per cent. died of the disease. He further found that women delivered at their own homes hardly ever died from that disease, and that, moreover, in those maternity hospitals where no medical students were taught, the puerperal mortality rate was less than in the others, with the exception of the Paris Maternité, where post-mortem examinations were made by midwives. He therefore assumed that the cause of the disease was some cadaveric material carried by the medical students, or others engaged in autopsies, to the lacerated genital tracts of the women. He further found that other circumstances such as overcrowding, fright, &c., had no influence on the incidence of the disease. It was, however, the death of his friend, Professor Kolletchka, from a post-mortem wound that turned his surmise into a certainty, for at the autopsy, which he attended, he found that the post-mortem lesions discovered in his dead colleague were identical with those constantly seen in the puerperal cases. Accordingly, he gave instructions that all medical students attending maternity cases should use chlorine water to disinfect their hands, and

[1] "Med. Hist. of Greek Anthology": *Proc. Roy. Soc. Med.*, 1914, vii (Sect. of Hist. of Med.), p. 33.

that their finger-nails be thoroughly cleaned before attending a labour. The result was dramatic, for in the following year, 1848, the maternal mortality from puerperal infection in the medical students' department fell from 11·4 per cent. to 1·27 per cent., i.e., to a lower figure than prevailed in the midwives' department! In order to prove his case up to the hilt, he showed by experiment that the transference of pus from women to puerperal animals was fatal to the latter. Semmelweiss, however, like Wendell Holmes, was, in spite of his persistency, unable to convert the obstetricians of his time, and he died of a broken heart and deranged mind.

In 1863 Pasteur discovered the microbic origin of infectious disease, and in 1875 Lister's methods of antisepsis (subsequently replaced by those of asepsis and surgical cleanliness for the prevention of bacterial growth) were applied to puerperal cases, and the mortality from child-bed fever fell from the 150-300 per 1,000 to about 2 per 1,000.

Notwithstanding, however, this remarkable reduction in maternal mortality as the result of the introduction of antiseptic and aseptic methods, the absolute number of deaths from puerperal septicæmia is still high. In 1923 close upon 1,000 women died from that cause in England and Wales alone—and yet these deaths, being due to microbic infection, were preventable and should therefore have been prevented. Indeed, we see from Table LXXXI that the mortality rate from this disease has been practically stationary since 1913.

Cause of Stationary Maternal Mortality Rate.—That the stationary rate of maternal mortality from puerperal sepsis is not entirely due to inefficient asepsis on the part of the accoucheur is proved by the fact that the condition occurs in all kinds of midwifery practice—whether rich or poor, town or country, private or hospital, and whether the cases have been attended by midwives or doctors. Indeed, cases of puerperal sepsis have occurred even in women who have delivered themselves without any vaginal examination or operative interference. Eden states that amongst 16,265 cases delivered inside the large lying-in hospitals during a number of years, there were 21 deaths from sepsis—a mortality of 1·20 per thousand. These deaths did not especially occur either in difficult forceps or other manipulation cases, nor had these cases been previously treated outside a hospital, so that no dirt had been introduced into them before admission into the hospital. Also, there has been no reduction

in the mortality since the Midwives Act, 1902, which made registration, instruction and supervision of midwives compulsory. It must be added, however, that, to some extent, the stationary character of the rate may be apparent only and due to improved certification of the causes of death, so that the real mortality rate may have fallen. In the United States, for instance, this improved certification has been responsible for an apparent rise in the rate.

Bacteriology of Vagina.—From what I have said it would appear, therefore, that a considerable proportion of the cases of puerperal sepsis occurs, not as the result of entrance of germs introduced by the manual manipulation of the accoucheur, but arises in some other way. A certain number of cases are due to infection of endogenous origin, i.e., arise from infection of the puerperal wound by organisms that had been lodging in the patient. The normal uterus contains no organisms, but bacteriological examination of the vagina of the pregnant woman has revealed the following organisms: streptococci, staphylococci, *Bacillus coli*, gonococci, pneumococci, and a few others. Most of the vaginal streptococci are of no great importance. Indeed, in no less than 66 per cent. of parturient women have streptococci been found, and yet puerperal septicæmia occurs in a comparatively very small number of cases. In about 1·3 per cent. of women the vagina contains hæmolytic streptococci, and it is these, as well as the gonoccoci and *B. coli*, which may give rise to septicæmia. In eighteen fatal cases, hæmolytic streptococci were found in the blood in sixteen. It is believed that these organisms are introduced during coitus. Blair Bell gives the following statistics with respect to coitus in 54 cases of labour:—

TABLE LXXXIV.

Date of last coitus	Total number of cases	Number of septic cases	
		Total	Per cent.
Within 7 days of labour	20	11	55·6
Between 7 and 21 days of labour ..	12	5	41·8
More than 28 days before labour ..	22	5	22·7

Similar results have been found by Lockhart.

These results afford a possible explanation of the considerable rise in mortality from puerperal sepsis in 1919 and 1920. The figures for these years, when plotted out in months, are found to correlate remarkably well with army demobilisation figures of nine months before, suggesting—but no more than suggesting—a

relationship between gonorrhœa in husbands and the incidence of puerperal sepsis in the wives.

Causes of Puerperal Sepsis.—It has been said that there are two things known for certain about puerperal sepsis; one is "nothing," and the other is "that the medical attendant will be blamed." Whilst the second thing is quite certain, the first is certainly an exaggeration. From what I said, we see that we may classify the causes of puerperal sepsis into (1) exogenous, i.e., those introduced from the outside; (2) endogenous, i.e., those residing in the woman herself. From this it follows that any examination or operation performed after rupture of membranes, however perfect may be the aseptic precautions, is attended with serious risk.

Exogenous causes include cases of infection from the hands and clothes of the accoucheurs, especially of those who have recently attended necropsies or dressed wounds; infection from the nose and throat of the doctor or midwife, who may be suffering from tonsillitis, rhinitis, &c., and also from infection by some "handy women" assisting at the confinement.

Endogenous cases may occur from the bacteria present in the vagina, or from an active remote focus in the patient, such as otitis media or septic finger, dental caries, &c. Statistics of Cæsarean sections published by Amand Routh, in 1911, and by Eardley Holland, in 1921, show that whilst the mortality of such operations before rupture of membranes is about 2 per cent., that occurring after rupture of membranes is about 10 per cent., suggesting that after rupture of membranes vaginal bacteria invade the uterus.

The following table represents the **maternal mortality** in England and Wales, 1921-23, **according to locality**:—

TABLE LXXXV.

	1921	1922	1923
County Boroughs—			
Sepsis	1·54	1·62	1·41
Total	3·94	3·90	4·01
Urban Districts—			
Sepsis	1·27	1·27	1·25
Total	4·03	3·96	3·92
Rural Districts—			
Sepsis	1·25	1·16	1·15
Total	4·22	4 01	3 87
All Districts—			
Sepsis	1·38	1·38	1·30
Total	3·91	3·81	3·81

From these figures we see that, although the total maternal mortality is higher in the rural than the urban areas, the mortality from sepsis is lower in the former than in the latter.

Dr. George Geddes,[1] who investigated the relative frequency of puerperal sepsis and of industrial accidents in different districts of Lancashire, believes that such accidents with the resulting septic wounds are, through the innocent and unconscious agency of the medical practitioner in attendance both upon the accident and the puerperal women, the source of sepsis in the latter. In support of his thesis he points out that during the cessation of work in the mining districts in the times of strike there is a fall in the incidence rate of puerperal sepsis. On the other hand, Caradog Jones, on passing Dr. Geddes' figures through the statistical mill, finds that the correlation coefficient between puerperal sepsis and accident rate, other than in mining districts, is, in view of the size of the probable error, probably insignificant. Thus, r between puerperal sepsis and accident rate per 1,000 population in mining districts $0{\cdot}216 \pm 0{\cdot}058$ for all districts, and $0{\cdot}110 \pm 0{\cdot}067$ for districts other than mining districts. There is, however, some significant correlation between puerperal sepsis rate and erysipelas rate, even in non-mining districts ($0{\cdot}217 \pm 0{\cdot}065$).

Dr. Geddes concludes as follows: "One cannot handle pitch and remain undefiled. It is equally true that one cannot treat septic wounds without risk of being contaminated. . . . The cause of puerperal sepsis does not depend upon either the professional proficiency of the medical practitioners or the service of midwives, but upon the conditions under which they have to practise midwifery."

In view of the insignificant value of r in the case of non-mining areas, it must be said that Dr. Geddes' thesis has not been substantiated.

Recent work has shown that the **prognosis** in cases of puerperal sepsis depends upon the number of organisms per cubic centimetre of blood. In cases of 1-10 cocci per cubic centimetre recovery is common, but if there are 50 per cubic centimetre it is very uncommon.

Notification of Puerperal Fever.—Under the Notification of Infectious Diseases Acts, puerperal fever is a notifiable disease, but there is no doubt that, for a number of reasons, notification is not complete, for in certain parts of the country the proportion of deaths from that disease to cases notified was 1,364 to 1,000! This means that only one case in about fifty has been notified.

The reasons for failure of notification are[2]:—

(1) The natural unwillingness on the part of the practitioner to suggest to the patient and her friends that her slight pyrexia, which might turn out perhaps to have been a mild case of influenza, may be a case of puerperal sepsis.

[1] "Puerperal Septicæmia." Bristol and London, 1926.

[2] See Discussion on "Notification of Puerperal Sepsis," *Proc. Roy. Soc. Med.* (Sect. Obst. and Gyn.), November 6, 1924.

(2) The idea widely prevalent that the doctor or midwife in attendance is necessarily responsible for the occurrence of the sepsis.

(3) Failure on the part of the authorities to provide the medical attendant with the necessary advice and hospital accommodation for the patient. If the Medical Officer of Health would in all such cases communicate with the doctor at once, offering consultation with a specialist free of charge to necessitous women, or removal of the patient to a hospital, and generally show evidence that the main object of the notification is a sympathetic desire to help the patient and the doctor, the main objection against notification would disappear. Indeed, in Birmingham, where sufficient hospital accommodation for such cases is available, there were about five and a half notifications to one death from puerperal sepsis in each of the years 1922 and 1923. Such provisions have now been authorized by the Public Health (Puerperal Fever and Puerperal Pyrexia) Regulations, 1926 (see below).

The total number of maternity institutions approved by the Ministry of Health during the year 1925 was 146, containing a total of 2,216 beds as follows: 79 voluntary institutions (1,347 beds), 67 municipal institutions (869 beds).

For the purposes of notification the following definition of puerperal sepsis was put forward by Sir W. J. Sinclair in 1905: "The term puerperal fever shall include all cases in which, within seven days after the birth of the child, alive or stillborn, the mother shall have a rise of temperature exceeding 100·4° F. (= 38° C.) with quick pulse, maintained for a period exceeding twenty-four hours, without any obvious cause other than the puerperal state. It shall also include all cases in which in the same period there has been the occurrence of rigor, with attendant illness, without any obvious cause other than the puerperal state."

The British Medical Association, in 1922, considered that all cases in which a temperature of over 100° F. occurred twice in twenty-four hours between the second and eighth days should be taken as cases of puerperal sepsis.

The Public Health (Notification of Puerperal Fever and Puerperal Pyrexia) Regulations, 1926, which came into operation on October 1, 1926, make also puerperal pyrexia, i.e., any febrile condition occurring in a woman within twenty-one days after childbirth or miscarriage, notifiable, and empower the Local Authorities to offer the medical attendant further medical

or bacteriological help, the service of a trained nurse, or the removal of the patient to a hospital.

Maternal Mortality from Causes other than Sepsis.—A glance at Table LXXX will show that we may divide these causes of death into the following groups, viz. :—

A. **Causes associated with Pregnancy.**
  (*a*) *Accidents*, e.g., abortion and miscarriage, ectopic gestation, &c.
  (*b*) *Diseases*, e.g., albuminuria, eclampsia and other toxæmias of pregnancy.

B. **Causes associated with Labour.**
  (*a*) *Accidents*, e.g., post-partum hæmorrhage, puerperal embolism, rupture of uterus, &c.
  (*b*) *Diseases*, e.g., puerperal insanity, pulmonary tuberculosis aggravated by childbirth, &c.

The Prevention of Puerperal Sepsis.—Puerperal sepsis may largely be prevented, and the following steps must be taken by every obstetrician or midwife in the prevention of this serious complication :—

(1) Careful and thorough ante-natal examination to detect : (*a*) abnormal presentation, (*b*) pelvic obstruction, (*c*) disproportion between size of pelvis and that of fœtus, (*d*) ill-health of woman (anæmia, or any other disease).

(2) Remove all possible sources of endogenous infection, viz., pyorrhœa, pharyngeal and nasal sepsis, chronic streptococcal vaginitis, chronic constipation, appendicitis, and bacteriuria, suppuration of ears, &c.

(3) Prevent access of infection from outside by observing the same aseptic precautions as at a surgical operation :—
  (*a*) The lying-in room must contain as little furniture in addition to the bed as possible, and should be thoroughly clean and free from dust.
  (*b*) The obstetrician must, in addition to thorough scrubbing of his hands, wear sterilised gloves and a sterilised overall, as well as a mask over mouth and nose. All his instruments, apparatus and dressings must be sterile.
  (*c*) The vulva and surrounding skin must be rendered aseptic with iodine, and the local hair shaved. In cases of suspected vaginal infection frequent

daily douchings should be carried out several days before labour. Also the anal area should be shut off from the vaginal area by means of sterile towels.

(*d*) No needless vaginal examination to be made, and any such examination to be as gentle as possible.

(*e*) If intra-uterine manipulations are necessary, an intra-uterine iodine or violet-green douche should be given. Do not hasten delivery by stretching the cervix.

(*f*) Leave the uterus empty, so as to help proper contraction of uterus, as well as to prevent decomposition of remains of the ovum. Also encourage sitting up in bed as soon as possible to help vaginal drainage.

(*g*) Avoid tissue bruising and tearing, by proper ante-natal methods, and repair tears immediately, if they occur.

(*h*) Avoid transference of infection from assistants to woman.

(*i*) Forbid coitus during last few weeks of pregnancy.

(4) Lastly, the parturient woman's condition of health must be in as good a state as possible, and any excessive bleeding, whether ante-, intra- or post-natal, must be carefully avoided, as hæmorrhage lowers the woman's resistance to infection.

Recent work has shown that, as a general rule, the *resistance* of a puerperal woman to streptococcal infection is raised rather than lowered (when measured by the leucocyte count as well as by the bactericidal power of the blood). Thus, counts of 20,000 per c.mm. are quite common during and immediately after labour. This is probably a provision of nature to protect women from puerperal infection.

On the other hand, in women suffering from puerperal sepsis the bactericidal power of the blood is greatly diminished. Thus, in a certain experiment the same number (390) of streptococci were implanted into equal volumes of blood from a septicæmic patient and of normal human blood. After incubation for two hours, the same volume of each of these infected bloods was mixed with malted agar, and the number of colonies grown were counted. The septicæmic blood grew 163 colonies (i.e., 72 per cent. of the cocci survived), whilst in the normal blood only 6 colonies (i.e., 1·5 per cent.) were grown (fig. 47).

It has further been shown that intramuscular injection of nuclein will double or treble the number of leucocytes in the blood within four or five hours, as well as increase the bactericidal power of the blood.

From these experimental results it follows logically that a step in the prevention of sepsis (in addition to the other points considered above) would be to take a leucocyte count in each case, either during or immediately after labour (especially in cases of difficult labour), and if the figure is below 12,000, to give an intramuscular injection of nuclein ($2\frac{1}{2}$ c.c. Parke, Davis and Co. Solution No. 1) to reinforce the patient's leucocytes ! [1]

RESULT OF A BACTERICIDAL TEST WITH HÆMOLYTIC STREPTOCOCCI.

Normal man's blood. Septicæmic blood.

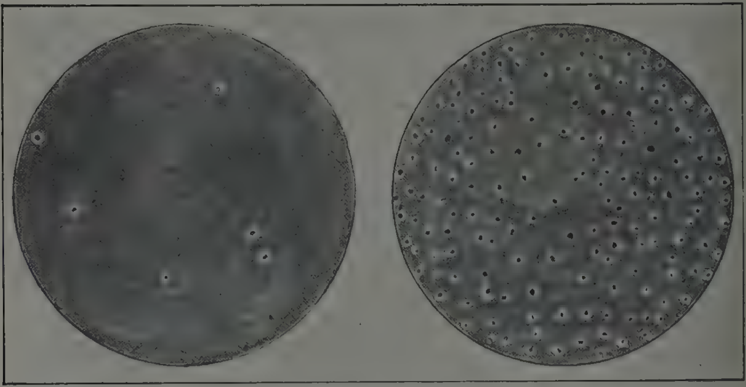

FIG. 47.—390 streptococci were implanted in each blood (L. Colebrook).

Prevention of Maternal Mortality in General.—Dame Janet M. Campbell, in her report to the Ministry of Health (Circular 517, Maternal Mortality, 1924), recommends the following measures for the prevention of maternal mortality :—

(1) An improvement in the quality of professional attendance —the doctor, midwife and maternity nurse.

(2) The provision, by the local Public Health Departments,

[1] See Leonard Colebrook, " Some Laboratory Investigations in Connexion with Puerperal Sepsis," *Proc. Roy. Soc. Med.* (Sect. Obst. and Gynæc.), Dec. 3, 1925.

of ante-natal centres, maternity beds, domiciliary midwifery, investigation of causes of death, &c.

(3) Social and educational measures of expectant mothers and their husbands through propaganda devised by the local authority, &c.

It is on these lines that the Maternity and Child Welfare Act, 1918, was framed as a necessary corollary to the Midwives Acts.

The results of efficient ante-natal supervision in preventing maternal mortality are well shown in the following tables for Huddersfield (England), and for Toronto (Canada):—

TABLE LXXXVI.—MATERNAL MORTALITY IN HUDDERSFIELD, 1916-1923 (Dr. S. G. MOORE, M.O.H., HUDDERSFIELD).

Year	Number of pregnancies notified	Number of deaths from pregnancy and parturition	Rate per 1,000 births	Number of pregnancies not notified	Number of deaths from pregnancy and parturition	Rate per 1,000 births
1916	215	1	4·65	1,691	9	5·32
1917	381	—	0·00	1,269	10	7·84
1918	506	1	1·97	1,069	6	5·61
1919	525	1	1·9	994	5	5 03
1920	807	1	1·25	1,295	6	4·61
1921	796	1	1·25	1,253	15	11·97
1922	559	3	5·36	1,268	7	5·52
1923	579	2	3·45	1,175	7	5·96
Total	4,368	10	2·291	10,012	65	6·49

TABLE LXXXVII.—RECORD IN OBSTETRICAL CASES, TORONTO GENERAL HOSPITAL, FOR TWO-YEAR PERIOD, TO SHOW THE INFLUENCE OF ANTE-NATAL SUPERVISION (J. G. FITZGERALD).

	Semi-private cases not supervised	Public ward cases not supervised	Public ward cases supervised
Number of cases ..	1,198	505	461
Deaths of mothers ..	10 (0·8 per cent.)	18 (3·5 per cent.)	2 (0·4 per cent.)
Eclampsia	20 (1·6 ,,)	16 (3·0 ,,)	2 (0·4 ,,)

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HOLMES, OLIVER WENDELL. "The Contagiousness of Puerperal Fever," 1873. Reprinted in C. N. B. Camac's "Epoch-making Contributions to Medicine," London, 1909.

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PART II

ANTE-NATAL HYGIENE

SECTION I.

THE ANTE-CONCEPTIONAL STAGE

"Destiny touches us with magical finger, long before consciousness awakens to the forces that have been set to work in our personality, launching us into the universe with country, forefather, and physical predispositions, all fixed without choice of ours."

MORLEY'S "Rousseau," i, 10.

CHAPTER VII

HEREDITY AND ENVIRONMENT IN RELATION TO CHILD HYGIENE

"I consider a human soul without education like marble in the quarry, which shows none of its inherent beauties until the skill of the polisher fetches out its colours, makes the surface shine and discovers every ornamental cloud, spot and vein that runs through the body of it. . . . What sculpture is to a block of marble education is to a human soul. The philosopher, the saint, or the hero, the wise, the good or the great man, very often lie hid and concealed in a plebeian, which a proper education might have disinterred and have brought to light."—ADDISON, *Spectator*.

Heredity is the force residing in the substance of the male and female germinal cells which determines that a person should bear some physical and mental resemblance to his parents or other relatives, immediate or remote. **Environment** means the surroundings to which an individual is exposed. These surroundings may be external, such as food, air, temperature, exercise, education, mental, spiritual and social, &c., or internal, viz., the chemical substances which are derived from the endocrine, or internal secretory glands.

"The Choice of Ancestors."—There are very few people who can really doubt that physical and mental as well as moral qualities are inherited. The best evidence of the relative importance of heredity and environment is that given by Galton in the case of identical twins (i.e., two children that have arisen from the division of a single egg). These twins, though living hundreds or thousands of miles apart, retain their initial resemblances of body and mind. They look alike, so that their closest relatives may be unable to distinguish them apart; their finger prints, though not identical are closely similar; their academic or business achievements are very similar, and even their thoughts as measured by association tests are marvellously alike. Binovular twins, on the other hand, even if living under the same environment, differ as much as ordinary brothers and sisters (figs. 48 and 49).

Advantage has been taken in the past of the similarity between identical twins to defeat the course of justice. The Midrash (sixth to ninth centuries) relates the story of a woman who, having been suspected by her husband of infidelity, was about to be tested by submission to the ordeal of bitter waters (Num. v. 28). Being guilty, she asked her twin sister, who closely resembled her, to impersonate her at the ordeal, and was of course found innocent. But, the story goes on, the guilty woman embracing her sister in gratitude for what she had done for her, smelled the bitter water and died on the spot, so that justice was vindicated!

FIG. 48.—Uniovular (identical) twins. Not only is their physical resemblance so much alike that it is very difficult to tell one from the other, but their mental achievements were very similar, as shown by the numbers of their school prizes, medals and other educational records. (From Clement Lucas's Bradshaw Lecture, "On Some Points in Heredity," London, Adlard, 1912.)

FIG. 49.—Binovular (ordinary) twins at the age of 7 years and 9 months. One is normal (height, 4 ft. 3¾ in.; weight, 4 st. 3 lb.), the other is an achondroplasiac dwarf (height, 2 ft. ¼ in.; weight, 2 st. 10½ lb.). There were two separate placentæ at birth. (Dr. Mary E. Weston.)

More recent and authenticated cases of the kind occurred in the English Law Courts. One such case was tried at the Oxford Circuit by Mr. Baron Jarrow. The judge had summed up strongly against the prisoner (accused of robbery), who had been identified by the prosecutor, when a man (who afterwards turned out to be the accused's twin brother working in collusion with him) suddenly rushed into court exclaiming that he had ridden fifty miles to save the life of the accused, who was innocent of the crime. The judge, jury and prosecutor admitted that they could not distinguish the stranger from the accused and acquitted the prisoner. When the intruder was indicted of the offence he pleaded "Not guilty," and pointed out that as

prosecutor had already sworn on the previous day to the other man, he could not logically swear to him now. As it was impossible to be sure who really was the guilty party, the second man also escaped the gallows (robbery having been then a capital offence). Such fraudulent miscarriages of justice would, however, be more difficult at the present time, because notwithstanding their close similarity in all other respects it has been found that the *finger prints* of identical twins are *not identical.*

It is obvious, therefore, that we are not in any sense born equal, and however right it may be to afford everybody equal opportunities, it is utter nonsense to say that they would or could all make equal use of them. Whilst, however, it is impossible to make sure of success in life, one may ensure the best chances of success by choosing one's ancestors.

The Pilgrim Fathers, who went over to America in the *Mayflower*, represented the best type of middle-class Englishmen, and half of America's greatest men are descended from twenty-three of these pilgrims. The following are a few of these pilgrims' descendants : six Presidents of the United States—John Adams, John Quincey Adams, James Garfield, Ulysses Grant, William Taft and Zacchary Taylor. Other great names are Emerson, the philosopher, Longfellow, the poet, Elihu Root, Daniel Webster, William Cullen, Bryant, &c.

Examples of brilliant English families are the Darwins, Pollocks and Wordsworths. The German Bach family is a very instructive example. The first of this line of musical geniuses was a flute-playing miller of 1550. His son, Hans Bach, was a violin player, his grandson, Heinrich, an organist. The subsequent generations contained a number of distinguished musicians, including John Christopher and John Sebastian.

On the other hand, the degenerate Jukes and Kallikak families (the names are fictitious) are well known as examples of bad stock to all students of eugenics, but the following case from Indiana, U.S.A., is very instructive. A feeble-minded woman, "Polly," had eleven illegitimate children, each with a different father. One of these, also feeble-minded, gave birth to eight illegitimate children, of whom seven were feeble-minded. Indeed, thirty-one out of fifty-six descendants of Polly have been feeble-minded.

Historical.—Ever since the beginning of recorded history thinkers have been speculating about the causes of the resemblances and differences between parents and offspring, although it is only within recent years that, as the result of intensive experimental and statistical research, the subject of heredity and eugenics can be said to have attained the rank of a science.

In very ancient times people looked upon heredity or the genetic relation-

ship between successive generations as being determined by the will of God, who visited the sins of the fathers upon their children even unto the third or fourth generation (Exod. xx. 5). The Prophets Jeremiah and Ezekiel, however, recognised that the son shall neither suffer for his father's wrongdoing nor shall he be rewarded for his father's good deeds. "What mean ye," asks Ezekiel, "that ye use this proverb: the fathers have eaten sour grapes and the children's teeth are set on edge. . . . Ye shall not have occasion any more to use this proverb. . . . The soul that sinneth it shall die. . . . If a man be just and do that which is lawful and right. . . . If he beget a son that is a robber . . . he (the son) shall surely die. . . . If he (the son) beget a son that seeth his father's sins and does not such like . . . ; he shall not die for the iniquity of the father . . . the soul that sinneth it shall die" (Ezek. xvii; see also Jer. xxxi, 28). Paul (Rom. ix. 21) believing in the immediate action of the Creator's will, asks the question, "Hath not the potter power over the clay, of the same lump to make one vessel unto honour, and another unto dishonour?" The Greeks as well as the Talmudic Rabbis recognised, however, that the theory of the Divine direct causal immanence in man's lot is not altogether tenable. "If," says Euripides, "one were to yoke good with bad, no good offspring would be born; but if both parents are good they will bear noble children." Sophocles does not agree with this view, for his observations have rightly taught him that "sometimes a noble offspring does not spring from well-born parents, nor an evil child from useless parents." The Talmudic Rabbis, also, while recognising that "as the tree so is the fruit," and therefore recommending the choice of a daughter of a man of character for a wife, still were aware that one cannot always rely on a good pedigree. Theognis, the Greek poet who flourished in the sixth century B.C., cried out against mercenary marriages. "We look," says he, "for rams and horses of good stock and believe that good will come from good; yet a good man is willing to wed an evil daughter of an evil sire if he but give her much wealth, nor does a woman refuse to marry an evil husband who is rich. Marvel not that the stock of our folk is tarnished, for the good is mingling with the base." Similar sentiments are expressed in the Talmud. Theognis gives his views on the relative values of heredity and environment as follows: "No education can make the bad man good; no Æsculapius can cure the moral taint. Just as roses and hyacinths do not spring from squills, so from the slave woman no free child can be born." Plato and Aristotle advocated State intervention to secure the mating of the best with the best, and that their offspring be reared by the Government. Seneca, in Epistle xi, says: "That which is implanted and inborn can be toned down, but not overcome. . . . Whatever is assigned to us by the terms of our birth, and the blend of our constitution, will stick with us, no matter how hard, or how long the soul may have tried to master them." The children of the unfit were to be destroyed. In the seventeenth century Walter Harris ("De Morbis Acutis Infantum," 1698) wrote as follows: "The prolific seed often so rivets the morbid disposition into the fœtus, that it can never afterwards be removed by any Art or Industry whatever. But let those who prefer a strong, vigorous and healthy offspring before Money, take care to avoid epileptic, scrofulous and leprous Mothers."

Theories of Heredity.

(a) The theory which prevailed for about 2,000 years, till the beginning of the last century, regarding the genetic relationship

between successive generations was that of *preformation*. It was believed that every reproductive cell in the human body, whether male (spermatozoon) or female (ovum), contained within itself a complete, but very much reduced, miniature of the human adult organism (*homunculus*) into which it develops by a process of evolution, or unfolding of the parts, in the same way as a bud unfolds to form a flower. Seneca says: "In the semen is comprised the entire course of the future man, and the unborn babe has written within it the law of a beard and a hoary head. For the whole body and load of future years are already traced in delicate and obscure outline in its constitution." It is clear that the preformation theory necessarily involved the further assumption that the homunculus contained within its own germ cells, in still more minute form, the individuals of the third generation, and so on, *ad infinitum*, so that within the germ cells of the first human being there must have been stored away—encased within one another—all the human beings that would ever be born. Another name for the preformation theory is the *scatulation* or packing theory, and is referred to in the Talmud, where a Rabbi is recorded to have said that "the Messiah will not come until all the souls inside the human body will become exhausted," an idea which Swammerdam in the seventeenth century expressed also in almost identical words: "Exhaustis his ovis humæni generis finem adesse."

Notwithstanding the absurdities to which such a crude theory leads, such eminent people as Malpighi (the famous Italian anatomist), de Bouffon (the well-known French biologist), and Haller (the father of modern physiology) believed in it. Malpighi and de Bouffon went as far as to assert that they actually saw the chick inside the unincubated egg, and Haller went to the trouble of calculating the number of human beings that God put away in Eve's ovary on the sixth day of creation. The German philosopher and mathematician, Leibnitz, applied this theory to the development of the soul, for he says that the "souls of men are present in the seed, like those of other species, in such wise that they existed in our ancestors as far back as Adam, or from the beginning of the world, in the forms of organised bodies." The theory was definitely shown to be untrue by Caspar Friedrich Wolff, the son of a Berlin tailor, in 1759, but it was not till the early years of the nineteenth century that it was finally abandoned.

(*b*) The next theory was that of *pangenesis*, formulated by

Darwin and Herbert Spencer. According to this, every cell in the body was believed to give off little particles (gemmules) which, circulating in the blood, come in contact with, and leave an impression upon, the germ cells, and thus the individual of the next generation inherits all the characters of its parents.

It is obvious that there are two corollaries which follow from this theory, viz.: (1) Any change taking place in the body as the result of some external environment (such as in A_2, fig. 50) must leave its mark on the germ cells, causing the same modification to be reproduced in the next generation (as in A_3). In other words, *acquired characters are inheritable* (Lamarckism). (2) The characters of the two parents blend in the children. This blending of characters led to the great statistical and mathematical work associated with the names of Francis Galton and Karl Pearson.

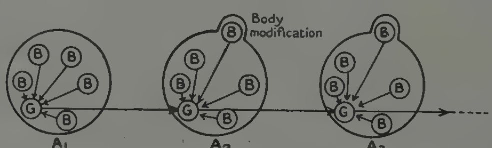


FIG. 50.—Diagram to illustrate the Pangenesis theory. B = body cells; G = germ plasm; A_1, A_2, A_3, = bodies of same organism.

The theory is plausible enough, except that there is no experimental evidence in its favour, and there is plenty of evidence against it, as we shall see later.

Galton's Experiment to Disprove Pangenesis.

Galton transfused the blood-vessels of one animal with the blood of another, and found, contrary to what one might expect from the gemmule theory, the offspring of the transfused animal not to exhibit any of the characters of the donor animal.

The experiment, however, does not prove or disprove anything, because transfused blood soon gets destroyed in the body, to be replaced by new blood formed by the receiving animal. Hence, the "many pretty wishes" to which such an experiment would give occasion, such as the "blood of a Quaker to be let into an Archbishop and such like," as Mr. Pepys expressed himself on hearing of a successful experiment on blood transfusion, must remain unsatisfied.

(*c*) The next great theory is that of Weismann (1883), and is called the theory of *germinal continuity*. It is based on exact observations on lower animals, in which it has been shown that when the fertilised ovum divides in its first cleavage into two

cells, one of these daughter cells goes to form the propagative or *germinal* part of the new individual and the other is destined to form its body or *soma*. The germinal part will again give rise to the germinal part as well as the body of the third generation, and so on, *ad infinitum*, as in the following diagram :—

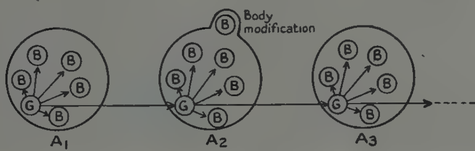


Fig. 51.—Diagram to illustrate the theory of Germinal Continuity. B = body cells ; G = germ plasm ; A_1, A_2, A_3, = bodies of same organism.

Hence any modification occurring in A_2 (fig. 51) as the result of some external environmental influence is not transmitted to the following generations A_3, &c., because such modification does not affect the germ plasm. In other words, *acquired characters cannot be inherited*. We shall return to the question of inheritance of acquired characters later (p. 242) ; in the meantime we can put the difference between Darwin's theory and that of Weismann as follows : According to Darwin it is the hen which gives rise to the egg, whilst according to Weismann it is the egg which gives rise to the hen. This, as we have seen, is a difference not without a distinction.

Proof in favour of Weismann's Theory.

Heape, in 1890, removed the fertilised egg from the body of a white long-haired rabbit and transferred it to the uterus of a rabbit with short grey hair. Normal development occurred, and an animal was born having all the characters of the real mother rather than of the foster-mother, showing that the characters of the developing embryo and fœtus had already been determined in the fertilised ovum, and that the changed intra-uterine environment failed to have any influence upon them. Still more striking is the result of Castle and Phillips, who in 1911 transplanted ovaries *before* fertilisation from one animal into another. Experience has shown that when an albino guinea-pig is mated with another albino guinea-pig the offspring are invariably albino. On the other hand, the offspring of an albino guinea-pig mated with a black guinea-pig are always black (fig. 52).

Castle and Phillips transplanted the ovaries of a black guinea-pig into the body of an albino guinea-pig whose own ovaries had been previously removed. The grafted albino animal was now mated with an albino male guinea-pig. The offspring were all black (see fig. 53) showing that the body or soma of the albino female animal had no influence on the ovary or germ plasm of the black animal.

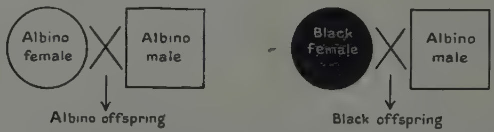


FIG. 52.—Result of crossing albino with albino and black respectively.

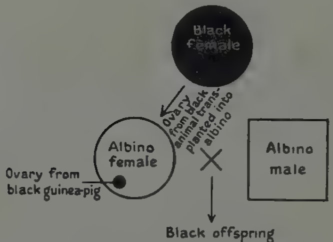


FIG. 53.—Castle and Phillips' experiment.

(*d*) *The Mendelian Theory.*—The theories which I have hitherto outlined concerned themselves only with an explanation of the resemblances between successive generations. Common experience, however, tells us that in many respects children differ fundamentally from their parents. For instance, the child of two brown-eyed parents may have blue eyes. Pasteur, probably the greatest man France has ever produced, was the son of two very ordinary parents, and the great English chemist and physicist, Faraday, the son of a blacksmith and a farmer's daughter. Leonardo da Vinci, philosopher, musician, sculptor, painter and universal genius of the fifteenth century, was the illegitimate son of a Florentine lawyer by a peasant woman; Isaac Newton was the son of an ordinary farmer; Dalton's father was a weaver; the father of the famous painter, Turner, was a barber, and Abraham Lincoln was the son of an illiterate father. Conversely we may find that two perfectly normal

parents have a feeble-minded child. The Mendelian theory, which has the great merit of being based upon the solid foundation of numerous breeding experiments in plants and animals, as well as of abundant statistical observations in man, offers a simple and ingenious explanation of these as well as of other phenomena of heredity. Mendel's original researches were carried out on peas, but it will be simpler for our purpose to choose an example from the animal world.

(i) On mating a black guinea-pig of pure race (i.e., one whose ancestors for many generations were black) with a white (albino) guinea-pig, the offspring (called the first filial generation or F_1) are found to be all black—none being white or intermediate in colour between black and white (see fig. 52). "Black" may therefore be called a *dominant* character, because it prevails to the total exclusion of the opposite character—" white."

(ii) If now two of the black individuals in F_1 are mated together, it is found that their offspring (the second filial generation or F_2) consist of both black and white animals (again none of intermediate colour), but the two kinds are in the proportion of three blacks to one white. The character "white" which was suppressed, or receded from view, in F_1, but reappeared in F_2, is, therefore, called a *recessive*, and the black animals in F_1 are called *impure dominants* (or impure blacks) or *heterozygotes*. The following (fig. 54) is a diagrammatic representation of the experiments :—

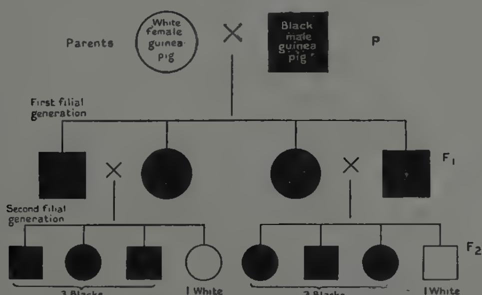


FIG. 54.—Diagram to illustrate the Mendelian mode of heredity.

Explanation of the Results.

Mendel's explanation of results such as these is very simple and ingenious. The male or female sexual cells of the black parent (P) are assumed to contain within them the factor which

determines the dominant character "black" (represented hereafter by B), and those of the white parent the factor which determines the recessive character "white" (W). In the case of F_1, however, whilst the body cells contain both B and W (although W recedes from view), their sexual cells consist of equal numbers containing B and W respectively, there being no single sexual cells containing both B and W. Thus:—

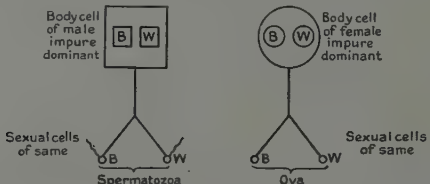


FIG. 55.—Segregation of character in the sexual cells.

When mating occurs between male and female animals in F_1 the following events may happen:—

(1) A "B-bearing" spermatozoon fertilising a B-bearing ovum. Result = Black animal (B B).

(2) A "B-bearing spermatozoon fertilising a W-bearing ovum. Result = Black animal (B W).

(3) A "W-bearing" spermatozoon fertilising a B-bearing ovum. Result = Black animal (B W).

(4) A "W-bearing" spermatozoon fertilising a W-bearing ovum. Result = White animal (W W).

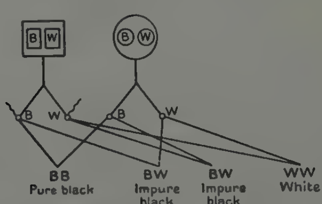


FIG. 56.—Explanation of Mendelian ratio of three blacks to one white in F_2, or one pure black (BB), 2 impure blacks (BW) and one white (WW).

Hence the animals in F_2 will consist of three blacks and one white (i.e., one pure dominant, two impure dominants, one recessive).

It is this segregation of contrasted characters (viz., dominant and recessive, in our case black and white) into separate sexual

cells, which is the main essence of the Mendelian theory, and the 1 : 2 : 1 ratio is called the Mendelian law.

Incomplete Dominance.—Dominance is not always complete, so that the impure dominant, instead of exhibiting the dominant character, may sometimes show a kind of blend. Thus it has been shown that the blue Andalusian fowl, well known to fowl fanciers, is really a heterozygote (F_1), the result of a cross between a black and a white-splashed fowl (i.e., white with a few black splashes), and that when they are bred amongst themselves the chickens in F_2 consist of black, blue and white-splashed varieties in the proportion of 1 : 2 : 1, in accordance with the Mendelian law. Hence the breeder can get more blues by mating the blacks and whites (the so-called "wasters" which he used to destroy before he knew the hereditary constitution of the blue fowl) than by breeding from the blues themselves. (See further, under Feeble-mindedness.)

Delayed Dominance.—Sometimes dominance does not show itself in heterozygotes until late in life. A good example is eye-colour, which is nearly always blue in babies and may change to brown later. We shall consider such cases in connection with Eugenics (p. 229).

Lucretius, in "De Rerum Natura,"[1] foreshadows Mendel's hypothesis as follows: "Sometimes, too, the children may spring up like their grandfathers, and often resemble the forms of their grandfathers' fathers, because the parents often keep concealed in their bodies many first beginnings mixed in many ways which, first proceeding from the original stock, one father hands down to the next father; and from these Venus produces forms after a manifold chance and repeats not only the features but the voices and hair of their forefathers."

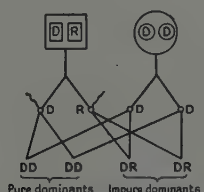


FIG. 57.—Offspring of cross between pure and impure dominants.

Corollaries of the Mendelian Theory.

(1) The mating of two pure dominants can only result in pure dominant offspring.

(2) The mating of two recessives can only result in recessive offspring.

[1] Quoted by F. W. Mott, "The Harveian Oration," 1925.

(3) The mating of a pure dominant with a recessive can only result in impure dominant (complete or incomplete) offspring.

(4) The mating of an impure dominant—

(i) With a pure dominant must result in offspring, all of which are dominant (50 per cent. pure, 50 per cent. impure) thus (see fig. 57).

(ii) With a recessive must result in offspring 50 per cent. of which show the dominant character (impure dominants), and the other 50 per cent. are recessive, thus :—

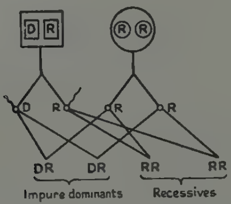


FIG. 58.—Offspring of cross between impure dominant and recessive.

(iii) With another impure dominant must result in pure dominants, impure dominants and recessive offspring in the proportion of 1 : 2 : 1 (see above, fig. 56).

All these theoretical expectations have been found on experiment to be true.

APPLICATION OF THE MENDELIAN THEORY TO CHILD WELFARE.

(A) **Eugenics**, from its etymology, means the science of being well born and is a term first introduced by Francis Galton, the cousin of Charles Darwin, in 1883, to denote "the study of the agencies under social control that may improve or impair the racial qualities of future generations either physically or mentally."

Although Galton was the founder of the modern eugenic movement, yet the suggestion that the human race might be improved by the method of selective breeding is very old and was known to the Greek, Roman and ancient Hebrew philosophers. When Abraham instructed his servant not to choose a wife for Isaac from among the daughters of the Canaanites he carried out a eugenic act. The same was the case when, in view of Rebekah's anxiety regarding a possible mésalliance between Jacob and one of the daughters of Heth, Isaac commanded Jacob to contract a consanguineous marriage in Padan-Aram rather than enter into an undesirable union at

home. The Greeks endeavoured to breed strong and healthy men and women. Plato, in the fifth century B.C., suggested State interference in marriage in order to ensure best with best, whilst the progeny of the inferior should be done away with. The same view was advocated by Aristotle.

Saleeby classifies eugenics into :—

(A) **Natural or Primary.**

(1) Positive.—The encouragement of worthy parenthood.
(2) Negative.—The discouragement of unworthy parenthood.
(3) Preventive.—The protection of parenthood from the racial poisons.

(B) **Nurtural or Secondary.**

(1) *Ante-natal.*—The treatment of syphilis of the expectant mother and her abstention from alcohol and other poisons.

(2) *Post-natal.*

(1) Infantile: breast feeding and avoidance of rickets.
(2) Home child nurture (1 to 5 years).
(3) School child hygiene.
(4) Hygiene of adolescence.

Statistical observations in man have shown that a very large number of characters—both normal and pathological—behave in a Mendelian fashion. Thus, whilst the children of parents each of whom has blue eyes are invariably blue-eyed, those of brown-eyed parents may have either brown or blue eyes, in the proportion demanded by the Mendelian theory, showing that brown is dominant to recessive blue. It is chiefly, however, the pathological or exceptional physiological characters which concern us here. It has been established that some of these characters behave like dominants and others like recessives. Thus hare-lip, cleft palate, glaucoma, diabetes insipidus, achondroplasia (a special kind of dwarfism in which the legs are short and the trunk is long), Huntington's chorea (a convulsive nervous disease, resembling St. Vitus' dance, occurring at about 30 to 40 years with dementia), &c., are dominants, whilst epilepsy, diabetes mellitus, feeble-mindedness, certain types of insanity, asthma, outstanding mental ability, great musical talent, albinism, a particular kind of deaf-mutism, &c., are recessives, as are also certain characteristics of the blood (in connection with "blood-grouping"), see p. 252.

TABLE LXXXVII.—STATISTICS SHOWING MENDELIAN MODE OF INHERITANCE OF FEEBLE-MINDEDNESS.

NF = normal heterozygote; FF = feeble-minded.

Case	Theoretical			Actual results found by Goddard			
				Number of cases		Proportion	
	One parent	Other parent	Offspring characters	Normal	Feeble-minded	Normal	Feeble-minded
1	NF	FF	Normal : feeble-minded : : 1 : 1	144	122	1·2	1
2	NF	NF	Normal : feeble-minded : : 3 : 1	83	39	2	1
3	FF	FF	All feeble-minded	6	470	1	78

The six normal children in the case of putative FF × FF may possibly be explainable by illegitimacy, where the father may have been normal.

It is, however, right to mention that Holmes and others do not agree that feeble-mindedness is a Mendelian character, although they admit that it is of an hereditary nature.

Classification of Mental Defectives.—Mental defectives may be classified into :—

(1) Mongols.

(2) Feeble-minded.

(*a*) Idiots.

(*b*) Imbeciles.

(*c*) Morons.

(*d*) Backward.

Mongols have a typical appearance which it is not necessary to describe in detail here. Suffice it to say that they have almond-shaped eyes obliquely set, squat nose, wiry hair and a transversely-fissured tongue. Their hands are broad and their fingers short. The condition is easily recognised early, i.e., in early infancy (at about 6-8 months). When such children grow up their mentality never exceeds that of a child of 7 years, and frequently remains at the level of a child of 4 years. The condition is supposed by some to be due to exhaustion of the uterus, because it generally occurs towards the end of the child-bearing period.

Feeble-mindedness is, as I have said, an hereditary Mendelian recessive character. The following standards define its various stages :—

An *idiot* is a person whose mentality does not exceed that of a child of 2 years.

An *imbecile* is one whose mentality does not rise higher than that of a child of 4 years.

A *moron* is one whose mentality does not advance beyond that of a child of 12 years.

A *mentally backward* person is one whose mentality is higher than that of a moron but less than that of a normal person.

Although feeble-mindedness may be acquired as the result of injury to or disease of the brain, the great majority of the cases, apart from mongols, are of hereditary origin. The following statistics, given by Goddard, show the recessive behaviour of feeble-mindedness: Out of 42 marriages between a heterozygote normal woman and a feeble-minded man there were 144 children, of whom 71 were feeble-minded and 73 were normal—almost exactly in the theoretical proportion of 1 : 1. Of 6 marriages between a feeble-minded woman and a normal but heterozygote man there were 193 children, of whom 122 were feeble-minded and 71 were normal. The discrepancy between expected and actual results in these cases may perhaps be explainable by illegitimate unions between the mothers and feeble-minded men.

The greatest danger of feeble-mindedness arises from the moron. This is so for two reasons, viz.: (1) Because on account of his high-grade defectiveness it is not easy to recognise him as a defective and take necessary precautions. (2) Because his mentality is sufficiently high to enable him to go about and get into all sorts of mischief. It has been definitely established that feeble-minded people increase twice as rapidly as the general population, and Goddard has estimated that a feeble-minded female is three times as great a menace to the community in the way of spreading the condition, as well as disease, as a feeble-minded male. The same authority estimates that at least 50 per cent. of the paupers and 50 per cent. of the prostitutes are feeble-minded.

Estimation of Feeble-mindedness.—According to Albert Binet, if children under 9 years of age show a backwardness of more than two years, or if children over 9 years old show a backwardness of more than three years they are probably feeble-minded. A more accurate method of recording the degree of backwardness is by using the **intelligence quotient** which is the ratio: $\frac{\text{Mental age}}{\text{Actual age}}$. If this ratio is greater than 0·80 the person is backward but not feeble-minded. If it is less than 0·75 the person is most probably feeble-minded.

Types of Psychological Tests for the Detection and Estimation of Mental Deficiency (Binet-Simon).

Sample tests for a child 3 years old.

(1) Ask the child to show its mouth, nose and eyes.

(2) Ask it to repeat two numbers which are not consecutive. (The child is first asked to repeat one number, then two, after pronouncing them distinctly at an interval of half a second. The test is repeated three times. If the child succeeds once out of the three times, it is considered as not below normal intelligence.)

FIG. 59.—Picture for the psychological testing of a child of 3 years From "Pédiatrie," by Sergent, Ribadeau-Dumas and Babonneix. Paris: A. Maloine et Fils, tome i, p. 230.

(3) Show it a simple picture illustrating some act, the interpretation of which should be easy, e.g., a dog saving a baby from drowning in a river lined with trees, and ask it to enumerate the objects seen in the picture (fig. 59). At the age of 3 years the child ought to be able to say that it can see a river, a dog, a baby, trees.

For a child 4 years old.

(1) Ask it whether it is a boy or a girl.

(2) Ask the child to name three familiar objects shown to it successively, e.g., a spoon, a pencil, a knife.

(3) Repeat three non-consecutive numbers (see 2, above).

(4) Show it two parallel lines of unequal length, say 2 and $2\frac{1}{2}$ in. respectively, separated at a distance of about 1 in. from each other, and ask whether it can tell which is the longer of the two.

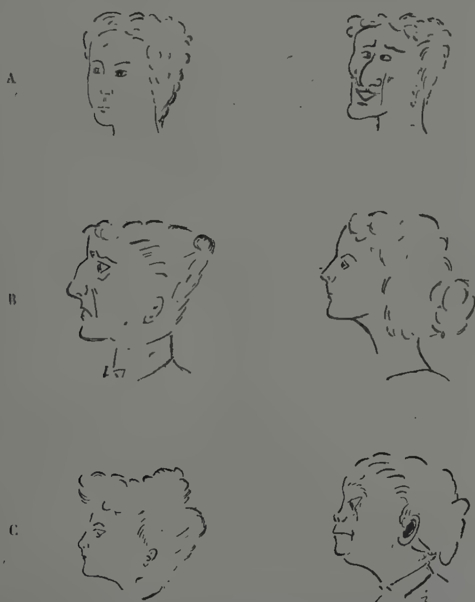


FIG. 60.—Test for a child of 6 years. From "Pédiatrie," by Sergent, Ribadeau-Dumas and Babonneix. Paris: A. Maloine et Fils, tome i, p. 233.

For a child 5 years old.

(1) Give it two objects (e.g., small boxes) looking exactly alike in shape and colour, and weighing say $\frac{1}{5}$ oz. and $\frac{3}{5}$ oz. respectively. Ask it to feel them and hold them up in its hands one after another and tell which is the heavier of the two. To make certain that a correct answer is not the result of a guess, one should repeat the experiment with two other similar objects

having different weights, but whose difference in weight is approximately the same, viz., $\frac{2}{5}$ oz.

(2) Ask it to count four similar objects (e.g., pennies) placed well apart from one another on a table.

For a child 6 *years old.*

(1) A child of this age ought to be able to tell whether any particular time of the day is morning or afternoon.

(2) It ought to be able to define the use of certain familiar objects such as of a fork, a chair, a knife, a table, &c.

(3) It ought to be able to distinguish distorted figures from regular ones of the same object, e.g., two faces. It is given a series of six faces arranged in three rows as follows: 1st row—left, normal face; right, same face distorted. 2nd row—right, normal face; left, same face distorted. 3rd row—left, normal face; right, same face distorted (fig. 60). A normal child of 6 years can tell the normal from the distorted in each row.

For a child 7 *years old.*

(1) It ought to be able to tell which is the right side of its body and which is the left. Thus, it ought to be able on command to place a finger of the left hand upon the right ear.

(2) A child of this age ought to be able to *explain the action* illustrated by such a simple picture as given in testing a child of 3 years (which latter should be able to enumerate the objects without interpreting the action).

(3) It ought to be able to execute three different actions for which the orders are given at the same time, e.g., "Put this key on that chair, then shut the door and then bring me the box which is on that other chair"—or some similar easy actions.

For a child 8 *years old.*

(1) It ought to be able to count from 20 to 0 backwards, as follows: 20, 19, 18 . . . 3, 2, 1, in twenty seconds.

(2) Show it pictures of persons in which some part of the body is missing, e.g., the eyes, the nose, the mouth, the ears, the arms, &c., and ask it to say, on being shown each picture in succession, what is missing in it (fig. 61).

(3) It ought to be able to give the date.

For a child 9 *years old.*

(1) It ought to know the various common coins of the realm.

(2) It ought to be able to enumerate the months.

For a child 10 *years old.*

(1) It ought to be able to copy a simple design from memory.

(2) It ought to be able to detect certain absurdities, e.g., you say to it that "the body of a man has been found cut up in twenty different pieces. It is believed that it was a case of suicide." A child of that age ought to be able to detect in twenty seconds (each) the absurdities in three out of five such statements.

For a child 12 *years old.*

(1) The child ought to be able to make a simple sentence containing three given words. (The examiner ought to give an

FIG. 61.—Picture from "Pédiatrie," by Sergent, Ribadeau-Dumas and Babonneix. Paris : A. Maloine et Fils, tome i, p. 235.

illustration of the kind of answer that is expected by making a sentence of three other given words.)

(2) It ought to be able to give the sense of a sentence in which the words are arranged in disorder, e.g., forbidden carriage it is this in to smoke.

For a child 15 *years old.*

(1) It ought to be able to find three rhymes to certain easily rhymable words, e.g., good (hood, food, mood), &c. The child ought to be given illustrations of what is meant.

(2) It ought to be able to solve simple problems, e.g., you ask him what would have happened in a certain house in which you saw the following people enter at short intervals of one another: a doctor, a lawyer, a priest.

To estimate the degree of mental backwardness, one submits the child of any given age to the tests proper for its age. If it fails to give the correct answers, one submits it to tests proper for a year, two years, three years, &c., younger, until it answers the tests correctly. Thus, supposing a child of 12 cannot answer higher tests than those for a child of 9, then its mental age is 9 years. It is, therefore, three years backwards and its intelligence quotient is: $\frac{\text{Mental age}}{\text{Actual age}} = \frac{9}{12} = 0{\cdot}75.$

As regards mental diseases, some are hereditary and others are acquired. Mott,[1] from a study of statistics based on a card system extending to 4,000 related individuals who have been in one of the London County Asylums, came to the conclusion that manic depressive insanity, or senile dementia, in an immediate or remote ancestor or collaterals may result in a condition of dementia præcox in the descendants. This was especially the case when the condition of insanity was present on both sides of the family. Mott therefore concludes that the marriage of people in whose family there is evidence of the existence of such mental diseases, especially on both sides, should be forbidden.

From the Mendelian corollaries enumerated on pp. 219 and 220 we can deduce the following **eugenic rules** :—

(1) If any particularly undesirable trait behaves like a dominant, then it is necessary for a person possessing it to abstain from procreation (Corollary 3), but unaffected members of the same family may marry other unaffected persons, whether of the same or a different family, with impunity. (Corollary 2.)

(2) If the undesirable trait is a recessive, then the person possessing it may marry a normal person in whose family there is no trace of the defect (i.e., a pure dominant). Their children will be normal (impure dominants), and may in turn marry normal persons in whose family there is no trace of the abnormality, i.e., pure dominants. (Corollary 4 (i)). Consanguineous marriages in such cases (even if both are normal) are, however, to be avoided, because each partner may happen to be an impure

[1] Frederick Mott, "Heredity in Relation to Mental Diseases," The Harveian Oration. *Brit. Med. Journ.*, October 24, 1925.

dominant, in which case 25 per cent. of the offspring will show the abnormality. (Corollary 4 (iii)).

So much for the straightforward rules which follow fairly obviously. But the matter is not so simple as this. Corollaries 1 and 4 above teach us that pure dominants can occur only under the three following conditions, viz. :—

(i) The mating of two pure dominants when all the children are pure dominants.

(ii) The mating of a pure dominant with an impure dominant, when 50 per cent. of the children will be pure dominants (the other 50 per cent. being impure dominants).

(iii) The mating of two impure dominants, when 25 per cent. of the children will be pure dominants.

As it is unlikely that a person with any abnormality, such as hare-lip, will marry somebody with a similar abnormality, it is clear that most of the people possessing dominant abnormalities are probably heterozygotes or impure dominants. Let us now take Huntington's chorea as an example of dominant character which is of eugenic importance. As the person suffering from it is most probably a heterozygote, 50 per cent. of his or her children will remain perfectly normal, and the other 50 per cent. will develop the disease at the age of about 35 or 40 years, when most people are already married and the parents of children. What is one to do with the children of such choreics? We have at present no certain means of telling which are the heterozygotes that will certainly develop the disease and transmit it to 50 per cent. of their children, and which are the recessives that are going to remain perfectly normal and have normal children. To forbid marriage to all would be unfair to 50 per cent. of them, whilst to allow them all to marry is obviously an injustice to society. Hence, before it is possible to formulate a fair eugenic rule in this case or in all other cases of *delayed dominance*, like glaucoma, &c., it is necessary to have some method of detecting such heterozygotes before the disease manifests itself. This is a lack which it is to be hoped will be supplied in the future.

Now take the case of such a recessive abnormality as feeble-mindedness. The eugenic rule is perfectly clear with respect to a person showing this trait; he or she must certainly not mate with a person similarly afflicted. But are we to allow such a person to have any children at all? It is true, as I have pointed out, that the results of the union between such a person and a

normal partner will be normal children—but this will only be the case if the normal partner is a pure dominant. Should, however, the partner happen to be a heterozygote, then by Corollary 4 (ii) half the offspring of such a union will be feeble-minded. Moreover, the union of a feeble-minded person with a purely dominant normal person results in normal children who are impure dominants, and whose union with similar heterozygotes would produce children of whom 25 per cent. would be feeble-minded, 50 per cent. would be normal heterozygotes again, and only 25 per cent. would be perfectly normal. In other words, such a union would only spread the condition subterraneously.

Hence we are faced with the following problem: Are we to segregate (or sterilise) all the feeble-minded and thus prevent them from multiplying? If we do, we shall certainly prevent the spread of feeble-mindedness, but we shall also prevent the birth of a large number of normal and possibly desirable individuals. Furthermore, such a measure, although it would stop the increase of this undesirable trait, would do nothing to stamp it out. It has been shown that whilst the large number of feeble-minded persons form only about 0·3 per cent. of the population, the number of normal carriers of feeble-mindedness (i.e., impure dominants) constitutes no less than 7 per cent. of the population. Intermarriage between such normal heterozygotes will result in progeny 25 per cent. of whom will be feeble-minded, and 50 per cent. will again be heterozygotes. It can be proved that by segregation (or sterilisation) of those actually affected it would take about 700 years to reduce the 3 per 1,000 to 1 per 1,000, and no less than 8,000 years to reduce the figure to 1 per 100,000 of the population! We see, therefore, that before we can usefully apply our eugenic rule to a case like this it would be necessary to have a method of detecting these heterozygote carriers. This we do not as yet possess, although it has been suggested that feeble-mindedness is *incompletely* recessive, and that certain psychological tests (like the Binet-Simon tests) may help to detect such heterozygotes. This is, however, a matter which requires thorough investigation and confirmation. Until we are provided with an absolutely reliable method of detection, the best way in which we can hope to reduce the large number of feeble-minded, from whom a large number of criminals, prostitutes and insane are recruited, is to have a **eugenic bureau** where the pedigree of every person is known for many generations, and to forbid a union between two heterozygotes.

The suggested Methods of Preventing the Propagation of Mental Defectives.

(1) **Education** of the general public in the great menace of feeble-mindedness, and the necessity of guarding against its spread. Such a method, though helpful, is obviously insufficient.

(2) **Legislation against the marriage of mental defectives** (or of persons in whose families there are strong tendencies to mental trouble) is being tried in certain States in America, such as Connecticut, Michigan, Minnesota, &c., but it is bound to fail as a *complete* preventive measure, since it does not prevent the illegitimate spread of this condition.

(3) **Segregation,** or **the isolation of feeble-minded into colonies where the two sexes are kept apart.** This, although a huge and expensive task, is not altogether an impracticable one, since not only will the expense be gradually diminishing every year, but the inmates can be made to become more or less self-supporting. The great objection, however, is the detection of the feeble-minded individual, for, as we have seen, not only is the higher grade defective difficult of being diagnosed, but the normal heterozygote carrier cannot even be suspected.

(4) **Sterilisation,** by means of vasectomy (i.e., severing the vas deferens, or the tube carrying the semen from the testicles to the urethra) in the male, or by salpingectomy (i.e., severing the Fallopian tubes carrying the ova from the ovaries to the uterus) in the female. The operation of vasectomy is exceedingly simple, although salpingectomy is somewhat more difficult. The advantage of this method is that it leaves the individual sexually potent but absolutely sterile. Such a law exists in Indiana and is applicable to every institution entrusted with the care of confirmed criminals, mental defectives, &c., although since 1913 it has, on account of the hostility it has aroused, been allowed to lapse.

In an elementary discussion like this, it is impossible to enter into a detailed consideration of **sex-linked characters** such as hæmophilia, colour blindness, &c., but it may be mentioned that the mode of transmission of such a character is as follows :—

(1) It only occurs in males. (2) If these marry females of normal stock, none of their children (i.e., none of F_1), whether male or female, will show the abnormality. (3) But whilst the male children of F_1 will never transmit the disease to their children, or children's children to any number of generations, (4) the females of F_1 will give birth to normal daughters and to sons

half of whom will be normal and the other half will possess the abnormality. The following diagram (fig. 62) will make it clear.

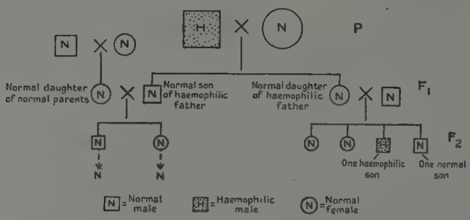


Fig. 62.—Mode of transmission of hæmophilia.

The eugenic rule in such a case is clearly this: All males, whether normal or affected, coming from such a stock may marry with impunity, whilst the normal females of such stock will transmit the affection to some of their sons. Here again, until we possess a reliable test for detecting the normal heterozygote female carriers of the disease, the only means of preventing the spread of the affection is a complete record of every woman's pedigree for a large number of generations.

(B) The Mendelian theory is also sometimes applicable to cases of **disputed paternity.** Supposing both the mother and putative father to possess a certain character which is known to behave as a recessive, then if the child does not possess that character it is practically certain that the man in question cannot be the father; e.g., if the child has brown eyes, and the mother as well as alleged father has blue eyes, it is almost certain that the latter cannot be the father. The same is the case if the man's and woman's blood belong, say, to Group IV and the child's blood belongs to another group.

The Relative Importance of Heredity and Environment in Child Hygiene.

Both heredity and environment play their parts in the health and welfare of a person, but the relative importance of each varies with different characters. Thus, the negro cannot change his skin, and the born imbecile can never be turned into a genius under any environment, because the colour of the skin in the one case, and the condition of the brain in the other, are purely hereditary characters over which environment has no influence at all. On the other hand, most infectious diseases are characters which are almost purely environmental. Thus, most people will

get influenza or small-pox when exposed to the disease, however strong and healthy they, or the families to which they belong, may be, unless they happen to be protected either by a previous attack or by vaccination. There are, however, a number of other conditions which depend both on heredity and environment. In some cases heredity has the greater influence, in other cases environment is the most important factor. For example, epilepsy and certain cases of asthma and insanity, &c., run in families, and although environment (such as food, mental anxiety, climate, &c.) does have a definite influence upon these diseases, its influence is small compared with that of heredity. The same applies to musical or mathematical ability. Most people can learn elementary arithmetic or to play the piano, but brilliant mathematicians and musicians are born and not made. Tuberculosis or consumption, on the other hand, is a disease which though to a certain extent hereditary, in the sense that the predisposition to the disease is inherited, is greatly influenced by environment, so that people with a family history of the disease, if exposed to proper surroundings as regards occupation, food, air, sunshine, &c., need not get the disease, or can be cured when they do get it. We may say that the physical and mental characters of every living being are to be regarded as, so to speak, the resultant of the two component forces, heredity or nature and environment or nurture. In some cases, such as congenital imbecility, nature is all powerful, and Shakespeare rightly made Prospero speak of Caliban as "a born devil on whose nature nurture will never stick"; in other cases environment is the most important factor. But in many cases both are of equal importance, and each is complementary to the other.

Heredity versus *Environment in Tuberculosis.*

A question over which controversy is raging is the mode of transmission of tuberculosis from parent to child. Clinicians and experimental pathologists insist that heredity is of no importance, and that if you can remove the children of tuberculous parents from their surroundings immediately after birth, they stand no greater chance of developing the disease than the children of non-tuberculous parents. On the other hand, statistical evidence points in the other direction, viz., that without an hereditary tendency there can be no consumption, since it has been shown that whilst the degree of "resemblance" (as measured by the correlation coefficient) for consumption between parents

and offspring is of the same order of magnitude as for certain hereditary characters such as height, eye-colour, &c., between parent and child, the "resemblance" for consumption between husband and wife, between whom the opportunities for infection are as great as between parent and child, is the same as for non-infectious characters, such as height, eye-colour, insanity, between the same people. The truth is, no doubt, between the two extremes, i.e., whilst there is an hereditary predisposition to tuberculosis, under favourable conditions there is no need for the child to develop the disease. On the other hand, a sufficiently massive dose of the germ will infect even people who have no hereditary tendency.

Experimental Work in the Hereditary Predisposition to Tuberculosis.

Sewall Wright[1] cross-bred the brothers and sisters of twenty-three pairs of guinea-pigs of pure breed, and thus got five lines of guinea-pigs particularly resistant to inoculation with the tubercle bacillus. He was able to obtain a regular scale of variation in tubercular resistance by crossing the offspring among themselves and with other stocks, and the resistance factor acted as a Mendelian dominant and is thus apparently determined by the genetic composition of the animal itself.

Dr. A. Govaerts, of Brussels,[2] studied the influence of parental tuberculosis on children by charting the weights of such children from birth up to the age of about 2 years alongside the mean chart of normal babies, and he found the following results :—

(1) In the offspring of healthy parents living under favourable environments the growth curve is parallel to the standard curve, and the mode of the dispersion (at 6 months) approaches the diagonal.

(2) In the offspring of healthy parents living in unfavourable environments the scatter of weight is narrower and is massed round the normal curve. Many children fall below the normal curve.

(3) In the offspring of tuberculous parents living under healthy conditions the majority of weights fall below the normal. From birth to 6 months all the weights are below the normal.

(4) Out of the twenty-one cases in each of the three groups, the following was the incidence of mortality during first year.

1st group	..	8	contracted illness	= 35	per cent.
2nd ,,	..	9	,, ,,	= 43	,,
3rd ,,	..	23	,, ,,	= 104	,, (i.e., some children have been ill twice).

The environmental influences having been the same in all the three groups, the differences can only be due to hereditary influences.

Congenital Transmission.—The reader must beware of confusing hereditary transmission with congenital transmission.

[1] "The Effects of In-breeding and Cross-breeding Pigs." U.S.A. Department of Agriculture, Washington. 1922.

[2] *The Eugenics Review*, April, 1925.

When we speak of a disease being transmitted by heredity we understand a condition transmitted from parent to offspring as the result of some physical condition residing in the germ-plasm of the parental sex cell. By congenital transmission, on the other hand, is to be understood that transference from parent to offspring, either as the result of some substance being carried as a foreign body by the parental sexual cells, e.g., in congenital syphilis, which may be acquired as the result of the **Spirochæta pallida** being carried by the spermatozoon, or as the result of some poison toxin or even microbes circulating in the maternal blood and affecting the developing fœtus, as happens, for instance, in the case of congenital small-pox, measles, or some other infectious diseases. Hence the student will understand that it is a misnomer to speak of hereditary syphilis. The correct name for it is congenital syphilis.

In the same way as we have congenital transmission of disease, so we also have congenital transmission of immunity to disease. This immunity, however, is as a rule of very short duration.

Tuberculosis.—Whilst cases of the transmission of the tubercle bacillus from mother to fœtus through the placenta have been recorded, they are so extremely rare as not to be worth considering from the point of view of ante-natal hygiene. But a matter of statistical interest, from the point of view of ante-natal hygiene in connection with tuberculosis, is the relation between this disease in one or other parent and fertility.

"Are the tuberculous specifically more fertile than the non-tuberculous? And, if so, to what extent is the net mass effect of this higher specific fertility checked by the shorter span of life of the tuberculous?" Martin Kacprzak investigated this problem, and he comes to the following conclusions[1]:—

(1) Any kind of tuberculous mating gives a smaller total absolute number of pregnancies than a non-tuberculous, i.e., on the whole tuberculous parents add less to the bulk of the population than if they were unaffected with the disease.

(2) Tuberculosis of the mother has a greater influence on the mean number of pregnancies than tuberculosis in the father, statistically considered.

(3) The lower absolute number of offspring per completed tuberculous family, as compared with the non-tuberculous, is largely due to early death of the tuberculous; slightly to a greater number of abortions and miscarriages in the tuberculous (see Table XIX, p. 116).

(4) The fertility of marriage is enhanced by tuberculosis of either one of the consorts.

(5) The mating of a tuberculous male with a non-tuberculous female shows an increased fertility at all age-groups.

[1] "Tuberculosis and Fertility," *Amer. Journ. Hyg.*, vol. iv, 1924, 605.

(6) Tuberculous women married with non-tuberculous men show a higher fertility than the non-tuberculous in all age groups.

(7) The mating of both parents tuberculous seems to show a higher fertility than the mating of both non-tuberculous.

Consanguinity of Marriage.

Although the laws prohibiting marriage between very close relatives are as old as the Bible, their origin was probably religious or sentimental rather than strictly eugenic. The nearest blood relations that are allowed to marry in this country are first cousins, and although the frequency of such unions is not accurately known, Sir George Darwin, some fifty years ago, estimated by an ingenious indirect method that about 3 per cent. of the total number of marriages are those between first cousins, and that such consanguineous unions are three times as common amongst the aristocracy as among the poor classes of society.

The Eugenic Effects of Consanguineous Unions.—There are some who believe that cousin marriages may lead to diminished fertility, increased infant mortality, as well as physical and mental deterioration in the offspring, and on these alleged grounds advocate the prohibition of such marriages. Indeed, in a large number of States in America cousin marriages are forbidden by law. In Catholic countries they are, of course, forbidden except by special dispensation of the Head of the State.

The general impression regarding the harmfulness of consanguineous marriages is of comparatively recent date, and is based upon the *experiences of certain isolated communities.*

In the Bahama Islands, for instance, in the West Indies, there is a place called Hopetown, inhabited by a colony of white people which has grown from a number of immigrants who settled there a few years ago. Owing to their isolated position, and because of their eagerness to exclude black blood, frequent intermarriage has been taking place amongst them, and it is said that there is a large number of degenerates there which are attributed to the close intermarrying of the inhabitants.

Several other examples of similar isolated communities containing considerable proportions of idiots, drunkards, deaf-mutes, &c., are frequently cited, and indeed, when examined superficially, they seem to afford very strong evidence of the dysgenic effects of such unions.

If, however, we probe the matter a little more deeply, we find that the evidence is not so strong as it appears at first sight, since it is possible to cite other instances of similarly isolated

communities where close in-breeding has been practised for a number of generations, and where the results have been remarkably good. For instance, there is the community of Batz, on the coast of France, where amongst a population of some 3,000 people, all of whom have sprung from consanguineous unions, there is not a single case of degeneracy. The same is the case in Smith's Island, off the coast of Maryland, and also in Cape Cod.

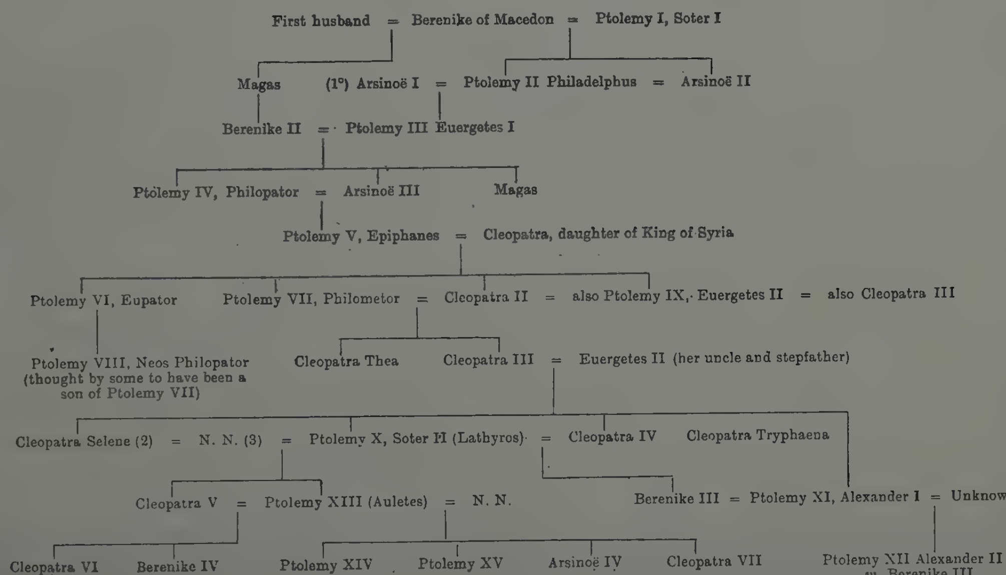
The evidence, therefore, being of a conflicting nature, we shall consider what history and biology can teach us.

Historical Evidence.—The Jews are probably the most virile race the world has ever seen, and yet a reference to the Bible will reveal the fact that they are descended from ancestors who continually intermarried among their very closest blood relations. Abraham married his half-sister Sarah, and their son Isaac married Rebekah, who was his grand-niece. Their son Jacob married his two cousins, Leah and Rachel. Amongst the eight tribes that originated from these consanguineous unions, one fails to find any evidence of any pathological condition. Indeed, a large number of members of these families were people of very great eminence, and most of them were distinguished in their own spheres. Moses and Aaron, the two outstanding personalities in history, sprang from a marriage between Abram and his paternal aunt Jochebed.

In ancient Egypt, too, marriages between brother and sister and other close blood relations were very numerous. The eighteenth dynasty, for instance, which repeatedly contracted incestuous marriages, consisted of nine distinguished monarchs who not only reigned brilliantly but whose physique and features, as far as can be ascertained from their mummified bodies, masks and statues, were in all cases exceptionally fine, distinguished and handsome. The same applies to the nineteenth dynasty kings, but the history of the Ptolemies is of the greatest eugenic interest, since some of the kings in that dynasty came from consanguineous and others from non-consanguineous marriages, and still comparison between the two groups fails to reveal any physical or mental differences. There were no cases of idiocy or any other undesirable trait amongst the consanguineous descendants, their average duration of life was long, viz., sixty-four years, and they did not lack in mental vigour. Some of them indeed (e.g., Cleopatra) were singularly clever.

Experimental Evidence.—Numerous investigators have of recent years studied the question experimentally by "in-breeding"

TABLE LXXXIX.—THE PTOLEMAIC DYNASTY.



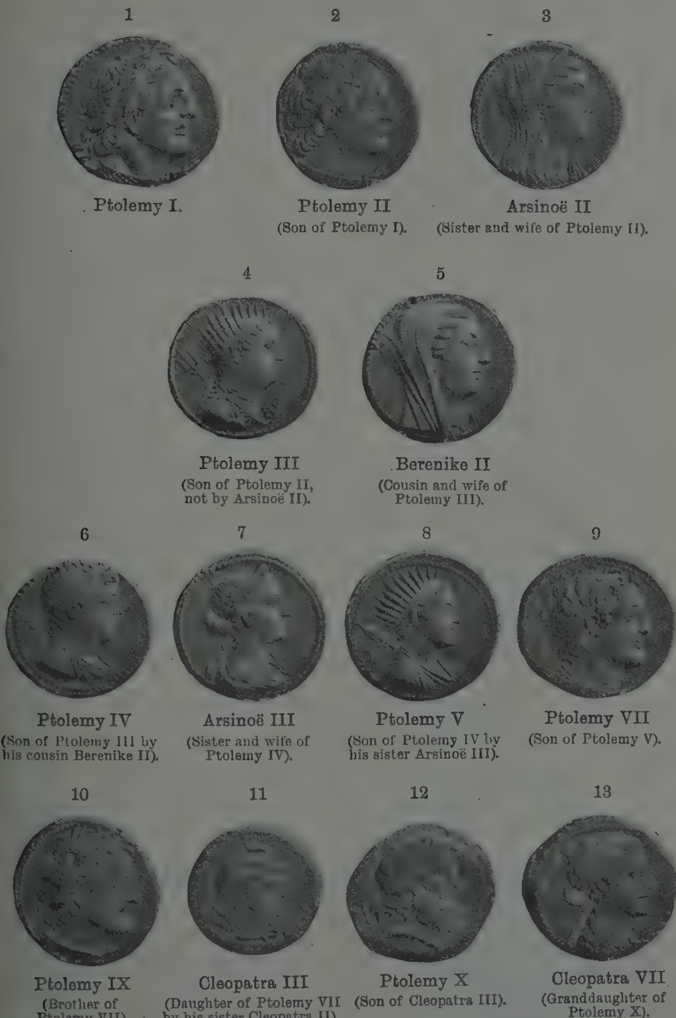


FIG. 63.—Ptolemaic dynasty. (Modified from paper by Marc Ruffer, on "The Physical Effects of Consanguineous Marriages," in *Proceedings of the Royal Society of Medicine*, Section of the History of Medicine, vol. xii, 1919.)

animals for many generations, and failed to detect any evil results, e.g., brother and sister mating has been carried on experimentally for twenty generations without bad effects on the offspring. Indeed they found that close "in-breeding" is very useful in perpetuating and developing any useful character. On the other hand, if there is any pathological character in the parents, similar "in-breeding" will also tend to perpetuate this; the same no doubt holds good in man. Thus, the numerous family of the Bachs freely intermarried consanguineously and were very prolific in musical talent.

The Wedgwood-Darwin families also contain several brilliant persons who are the offspring of consanguineous unions (see fig. 64).

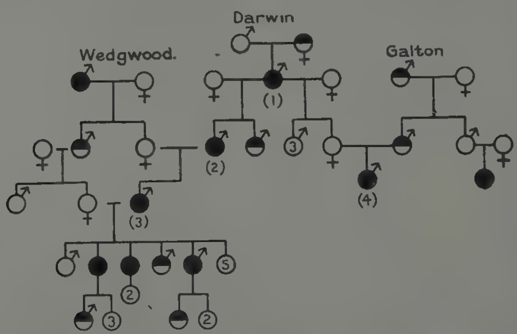


Fig. 64.—Pedigrees of the Darwin-Galton-Wedgwood families showing the good results of in-breeding in the case of clever families. ◓ = Person of great ability. ● = Fellow of Royal Society. The number inside a circle indicates the number of other children. The following are the names of those having numbers underneath them. (1) Erasmus Darwin; (2) Robert Waring; (3) Charles Darwin; (4) Francis Galton. Three sons of the marriage of Charles Darwin with his cousin were Fellows of the Royal Society and one is a man of great ability.

On the other hand, we also know that certain pathological characters like epilepsy, mental deficiency, retinitis pigmentosa, Friedreich's disease, certain paralytic and mental diseases, &c., when present in the families of both parents, show themselves with greater certainty in the offspring of consanguineous unions. Hæmophilia, for instance, has dogged the footsteps of almost every royal family in Europe on account of the great frequency of consanguineous marriages among them. Indeed, since the time when the mode of transmission of some of these morbid traits has become elucidated, the matrimonial affairs of members of the

various royal families have ceased to be the diplomatic concern in which the Foreign Offices of the respective countries are alone concerned; they have become problems in the solution of which the co-operation of the expert biologist is also called in.

To summarise: I think I am justified in saying that the study of the historical, statistical, and biological evidence at present available permits one to draw the following conclusions:—

(1) There is no evidence that a consanguineous marriage is followed by reduced fertility, or increased mortality or physical and mental degeneration in the offspring.

(2) There is nothing connected with consanguinity, *per se*, which makes a union between relatives prejudicial to the offspring. In other words, no morbid conditions will be created in the offspring of a cousin marriage if the parents are themselves of good stock.

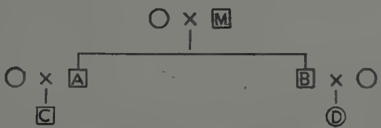


Fig. 65.—Inheritable character latent in each partner of a cousin marriage. The consanguineous union between C and D is: (1) Desirable if M = desirable trait; (2) Undesirable if M = undesirable trait.

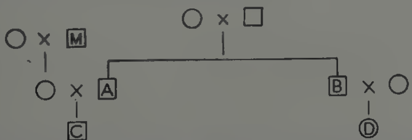


Fig. 66.—Inheritable character latent in one partner only of a cousin marriage. There is no more risk in the consanguineous union of C and D (although C's grandfather possessed some undesirable trait) than for C to marry outside the family.

(3) If, however, there be any tendency to certain diseases, which are known to be hereditary, in the family, then the risk of the disease appearing in the offspring is greatly accentuated by a consanguineous marriage, even if neither of the partners manifests the disease.

(4) On the other hand, if there is any particularly desirable character in the family, such as great musical or mathematical ability, or other mental or physical talent, then a cousin marriage in virtue of its tendency to perpetuate and fix that character in

the offspring, is much more desirable than a non-consanguineous union with a stranger, whose antecedents may be less well known.

(5) It should be added that consanguineous marriages should only be encouraged or discouraged if the character which it is desired to be perpetuated or eliminated lies latent in *each* of the two partners. If, however, it is latent in only one of the partners, then a marriage between cousins is neither more nor less desirable than a non-consanguineous union (see figs. 65 and 66).

The Inheritance of Acquired Characters.

We have seen above that characters which are not preformed in the germ (blastogenic), but which arise only through special influences affecting the body (somatogenic), are not inherited, e.g., no Jewish child has yet been born circumcised, and no girl has yet been born with pierced ears, in spite of the fact that these practices have been carried out for thousands of years. It is possible, however, for somatogenic characters to be converted into blastogenic ones. Thus, when any modification arising in the body as the result of some external environmental influence gives rise to the formation of some chemical substance which can circulate in the blood and reach the germ-plasm (e.g., by modifying the hormones or internal secretions of certain endocrine glands), then we have a somatogenic character converted into a blastogenic one and it becomes inheritable.

It is a well-known fact that when a foreign protein is introduced into the blood of an animal there are chemical antibodies formed in the blood which can destroy that particular protein. Messrs. Guyer and Smith injected finely ground lenses (of rabbit's eyes), suspended in normal saline, into the veins of a fowl. The serum of the fowl, which became capable of destroying rabbits' lenses, was then injected into the veins of a pregnant rabbit, when it was found that not only were the lenses in the embryo rabbits dissolved, but that the condition was intensified in the six succeeding generations on which observation was kept. Moreover, similar results were obtained when the male parent was treated in the same way, showing that the condition was due to an actual alteration of the germ-plasm rather than to transmission of antibodies from the mother to the fœtus by means of the placental circulation. Similarly, poisons such as alcohol and syphilitic poison circulating in the blood may affect the germ-cells and be thus transmitted to the next generation. Pavlov in 1922 found that conditioned reflexes are inherited and intensified in subsequent generations.

Agnes Bluhm, for instance, has shown that alcohol has a **selective action on the male germinal cells.** Recent work has shown that spermatozoa are of two kinds, viz., male determining and female determining, so that the sex of the child depends upon the kind of spermatozoon which fertilised the ovum. Bluhm finds that whilst the average normal sex ratio in white mice is 79 ♂ : 100 ♀, that of mice in which the males had been injected subcutaneously with alcohol was 122 ♂ : 100 ♀. She therefore believes that the vitality of the female-determining spermatozoa is more diminished by alcohol than that of the other kind.[1]

The selective action of alcohol upon germ-cells has also been shown by Danforth.[2] He finds that the percentage of Mendelian expectation of certain characters (but not of others) is affected by alcohol, as the following table shows :—

TABLE XC.

Experiment		Brachydactylia		Polydactylia		White	
Number	Part	Observed	Expected	Observed	Expected	Observed	Expected
1	A	$46{\cdot}7 \pm 3{\cdot}1$	$39 \pm 3{\cdot}0$	$39{\cdot}4 \pm 2{\cdot}7$	$36 \pm 2{\cdot}9$	100	100
	C	$39{\cdot}2 \pm 2{\cdot}5$	$39 \pm 2{\cdot}5$	$33{\cdot}2 \pm 2{\cdot}1$	$36 \pm 2{\cdot}2$	100	100
2	A	$43{\cdot}3 \pm 3{\cdot}1$	$39 \pm 3{\cdot}0$	$37{\cdot}9 \pm 2{\cdot}9$	$36 \pm 2{\cdot}9$	$50 \pm 3{\cdot}2$	$50 \pm 3{\cdot}2$
	C	$33{\cdot}1 \pm 3{\cdot}7$	$39 \pm 3{\cdot}8$	$37{\cdot}0 \pm 3{\cdot}7$	$33 \pm 3{\cdot}7$	$52{\cdot}1 \pm 7{\cdot}0$	$50 \pm 3{\cdot}9$
3	A	$53{\cdot}6 \pm 3{\cdot}4$	$39 \pm 3{\cdot}3$	$52{\cdot}0 \pm 3{\cdot}4$	$36 \pm 3{\cdot}3$	$59{\cdot}9 \pm 3{\cdot}5$	$50 \pm 3{\cdot}6$
	C	$42{\cdot}7 \pm 3{\cdot}5$	$39 \pm 3{\cdot}5$	$40{\cdot}1 \pm 3{\cdot}5$	$36 \pm 3{\cdot}4$	$56{\cdot}5 \pm 3{\cdot}6$	$50 \pm 3{\cdot}7$
4	A	$56{\cdot}3 \pm 5{\cdot}9$	$39 \pm 5{\cdot}7$	—	—	$42{\cdot}8 \pm 6{\cdot}3$	$50 \pm 6{\cdot}4$
	C	$38{\cdot}4 \pm 2{\cdot}9$	$39 \pm 2{\cdot}9$	—	—	$53{\cdot}4 \pm 3{\cdot}1$	$50 \pm 3{\cdot}0$
Totals	A	$48{\cdot}2 \pm 1{\cdot}8$	$39 \pm 1{\cdot}7$	$36{\cdot}0 \pm 1{\cdot}7$	$36 \pm 1{\cdot}7$	$52{\cdot}7 \pm 2{\cdot}2$	$50 \pm 2{\cdot}2$
	C	$39{\cdot}0 \pm 1{\cdot}5$	$39 \pm 1{\cdot}5$	$36{\cdot}0 \pm 1{\cdot}6$	$36 \pm 1{\cdot}6$	$53{\cdot}9 \pm 2{\cdot}0$	$50 \pm 2{\cdot}1$

A = Alcoholised ; C = Control.

Hence there is a definite selective action with regard to brachydactylia (in the case of birds).

Bertholet[3] examined microscopically the testes and ovaries of many hundreds of normal and alcoholic subjects in Lausanne and found degenerative changes in 82 per cent. of the alcoholic cases. He concludes, therefore, that the reproductive glands are more susceptible to alcoholic intoxication than the other organs.

[1] *Sitzungsber. d. Preuss. Acad. d. Wissensch.* (*Math.-phys. Kl.*), 34, 1921; and *Arch. f. Rassen- und Gesellschaftsbiol.*, September 16, 1924.

[2] *Journ. Exper. Zool.*, vol. xxviii, 1919, pp. 385-412.

[3] "L'Influence d'Alcoolisme sur les Organes et sur les Glandes Reproductrices," 1913.

Kyrle and Schoffer, experimenting on dogs, found the same degenerative changes in the sexual glands of the alcoholised animals. The works of Stockard, Laitinen and others have already been referred to (see p. 112).

In these cases, however, it is not so much injury to the germ-cells that is at work as the actual carriage by the germ-cells of the poison as a passenger from one generation to another. It is for this reason that syphilis is, as a rule, not transmitted beyond the second generation.

The Relation between Heredity and Environment.

People obsessed with the great importance of heredity are apt to assign a very subordinate position to the influence of environment upon the welfare of the individual during the various stages of his development. Nevertheless, the rôle played by environment is by no means negligible. It is true that "roses and hyacinths do not spring from squills," but it is equally true that a hen's egg will not develop properly when incubated either at the wrong temperature or in the wrong position, and that a blow on the head may destroy the brain power of the greatest genius. The truth is that each is complementary to the other. It must be remembered that it is not our actual but potential personalities or characters that are predetermined in the germ-cells. Even those that assert that heredity is the all-important factor in tuberculosis do not claim that a baby is (except as a pathological curiosity) born with tubercle bacilli in its body; it is only a special predisposition to the onslaught of the germ that they believe to be inherited, so that in the absence of all possibilities for infection the child will remain free from tuberculosis even according to the views of the most ardent eugenist.

Environment cannot create new characters, but it can and does determine which of the potentialities that are inherited shall be developed and to what extent. During pre-natal life not only can poisons and toxins, such as syphilis, alcohol, lead, morphine, phosphorus, infectious diseases, &c., pass from the mother to the fœtus through the placental circulation, so that the mother must as far as possible be protected from these poisons, but we can actually treat certain such acquired diseases of the fœtus (especially syphilis) by administering the appropriate therapeutic agent to the mother (see pp. 109, 110, and 278). As for post-natal life, the whole of modern hygiene and therapeutics is based upon the influence of environment upon the welfare of the individual.

Eugenics in Relation to the Child Welfare Movement.

It is claimed by the strict eugenist that the whole campaign against infant mortality is wrong in principle, since a reduction in the mortality of infants under 1 year old means a saving of the lives of a number of weaklings, most of whom would die in the second, third or fourth years of life. Moreover, say they, even if some of these should manage to survive to adult age they will only become the parents of weaklings like themselves. This argument is baseless in the one case and unsound in the other. Statistics show that in those places where the infant mortality rate is high for the first year of life, there also is the mortality rate high in the case of children between 1 and 5 years old, and vice versa. If the first eugenic argument were correct we should expect the opposite condition to prevail. As regards the second argument, it must be realised that it is not always the strongest physique that is the world's greatest asset. Many brawny persons have very poor brains, while such people as R. L. Stevenson, Chopin, Darwin and numerous other people of eminence had very frail bodies. Nelson was weak as a child, and under unfavourable environment would almost certainly have succumbed. Newton was feeble and undersized at birth and his life was almost despaired of, and the famous French mathematician and philosopher D'Alembert was a puny foundling. Voltaire was a puny infant at birth and was not expected to survive. In spite of severe small-pox at the age of 29, as well as several other diseases, he lived to the age of 80 years. To carry such an argument (even if true) to its logical conclusion would mean that we should also allow every woman with a contracted pelvis to die in labour, rather than help her by suitable means, lest her survival will perpetuate the same abnormality in her children. To treat such mothers, and at the same time to allow the more weakly infants to die, is an argument strongly reminiscent of Mr. Punch's cartoon, "The Spartan Mother."

Heredity and Child Mortality.

Large numbers of data have been collected by Karl Pearson and Mary Beeton in this country, and the Genealogical Record Office of Washington, which tend to show that mortality during the early years of childhood varies with the age at death of the father or near relation. Thus, out of 340 families (with 2,259 children) in which the father's or uncle's age at death was 90 or more, the death-rate of children under 5 was 53 per 1,000 as

compared with 400 for the Registration Area of the U.S.A. in 1880. A mortality of 53 for the first four years of life is smaller than the present-day mortality for the first year of life alone. This population, from whom the figures were taken, mostly consisted of poor people, many of whom were ignorant, and none of whom had the benefit of attending infant welfare centres. In fact, according to Popenoe and Johnson, from whose work on "Applied Eugenics" the above figures were taken, the only advantage which those children had over others was good heredity. They had inherited exceptionally good constitutions. Karl Pearson and Mary Beeton have shown that not only is the tendency to long life (brachybiotry) inherited, but the tendency to die young is also inherited. Ploetz, in a study of child mortality in relation to the father's age at death in various royal families, found a similar correlation to exist, showing that the higher mortality rate amongst children whose parents died young is not due to lack of care or neglect brought about by the early death of the parent. Thus, Ploetz[1] gives the following figures for 3,210 children in royal families in Europe:—

TABLE XCI.

	Fathers' ages at death								
	16-25	26-35	36-45	46-55	56-65	66-75	76-85	86 up	All ages
Number of children	23	90	367	545	725	983	444	33	3,210
Number who died in first five years	12	29	115	171	200	254	105	1	887
Per cent. who died	52·5	32·2	31·3	31·4	27·6	25 8	23·6	3·0	27·6

In spite of these statistics, however, heredity must not be given an undue prominence and blind one to the great possibilities inherent in efficient environment, since, as we have seen, intensive infant welfare work has been instrumental in keeping babies alive and well that would otherwise have died.

Human Sex Determination.—It can be definitely stated that the sex of the future baby is determined at the time of conception, and that it is the spermatozoon and not the ovum which is the

[1] A. Ploetz, "Lebensdauer der Eltern und Kindersterblichkeit," *Arch. für Rassen- und Gesellschafts Biologie*, vol. vi (1909), pp. 33-73.

determining factor. We have seen on p. 243 that these two types of spermatozoa—male and female determiners—are differently susceptible to chemical agents. They are also differently affected by physical factors, such as heat, X-rays, radium, &c., and they are known to swim at different rates. Hence it may be possible to find some method by which to separate the two types of sperm cells from each other and to produce one or other sex at will by artificially inseminating with the proper spermatozoa. This, however, is a matter on which we have at present but very little information.

Pre-nuptial Clinics.—Whilst it is in the present state of our knowledge impossible to make any far-reaching statements as to who should and who should not be allowed by the State to marry and have children, we do know enough to say that no person who is either himself mentally deficient or has a strong family history of mental trouble should be allowed to marry. The same applies to people suffering from uncured venereal disease or active tuberculosis. Deliberate breach of such a rule should be made not only a penal offence but a sufficient reason for divorce.

But even those who would object against the compulsory prohibition of marriage to anybody (whatever his physical or mental disability may be), cannot reasonably object to the establishment of pre-nuptial clinics, where all young people could *voluntarily* apply for having their physical and mental fitness graded—as it is quite possible to do satisfactorily—by means of special tests. Certificates would be awarded to those placed in Classes I and II. The value of such certificates would soon become recognised.

From what I have said, the reader will appreciate the truth of the statement that "the proper time to begin treatment of many diseases is one hundred years before birth."

Summary.

If we summarise the most important matters dealt with in the preceding chapters, we arrive at the conclusion that a well-organised system of child welfare care should include :—

(1) *Ante-natal Care.*
  (*a*) Pre-nuptial clinic.
  (*b*) Ante-natal clinics.
  (*c*) Pre-maternity wards in hospitals.

(2) *Intra-natal Care.*—
  (*a*) Maternity beds at hospitals for those who have not the convenience to be delivered at home.

(*b*) Free supply of well-trained midwives or doctors to women who can be delivered at home and who cannot afford to pay for professional care.

(*c*) Home-helps, i.e., women to look after the mother, children and the home during the puerperium.

(3) *Post-natal Care.*

(*a*) Infant welfare centres. The value of these institutions has been disputed, but there is very little doubt that they have been a great factor in the reduction of infant mortality. The only reasonable objection that can be levelled against them is that the bringing together of many infants under the same roof helps to spread infectious diseases amongst them.

(*b*) Child welfare centres for children from 1-5 years.

(*c*) School care.

(4) *Propaganda and Education.*

(*a*) Health visitors, to visit the homes and inculcate the elementary principles of maternal and infant hygiene.

(*b*) Free illustrated lectures to adolescent boys and girls.

(*c*) The teaching of mothercraft at schools.

(*d*) Free lectures to parents (of both sexes).

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PART II

ANTE-NATAL HYGIENE

"Teach us what we shall do unto the child that shall be born."
Judges xiii, 8.

SECTION II

POST-CONCEPTIONAL OR INTRA-UTERINE STAGE

"The building of a ship is more wonderful than the launching of it on its first cruise; the sculpturing of a statue is a greater thing than the unveiling of it; and so the making and growth of the infant in the womb are more momentous than its entrance into life that follows birth."—J. W. BALLANTYNE.

CHAPTER VIII

THE PHYSIOLOGY OF THE FŒTUS

"In the dark womb where I began,
My mother's life made me a man,
Through all the months of human birth
Her beauty fed my common earth,
I cannot see, nor breathe, nor stir
But through the death of some of her."

JOHN MASEFIELD

ALTHOUGH the lacunæ in our knowledge of ante-natal physiology are both large and numerous, we have a vast amount of information of the functional activity of the fœtal organism. In this chapter I shall attempt to give a brief outline of a few of the more important points in the physiology of ante-natal life which will help us to understand the principles of fœtal hygiene.

Physiological Connection between Mother and Fœtus.

During the nine calendar months extending from the moment of conception till the baby emerges from the mother's body, it lives a sort of parasitic existence inside the mother's womb. In fact, the Psalmist's description of the idols of the heathen might equally well be applied to it. It has eyes but it sees not; it has ears but it hears not; and, it may be added, it has a mouth but it eats not, and lungs but it breathes not. Its kidneys and bowels, too, are in a dormant state, and all its various functions of nutrition, respiration and excretion are carried out for it by its mother.

The fœtus is tethered to its mother's womb by means of the navel string, or umbilical cord, through which the mother sends to her child pure blood, containing all the nourishment required by the fœtus, and the latter sends back to the mother along parallel channels in the same cord the various waste products manufactured in its system (fig. 67).

The whole vital economy of the fœtus therefore depends on a proper interchange of blood between the mother and itself. This

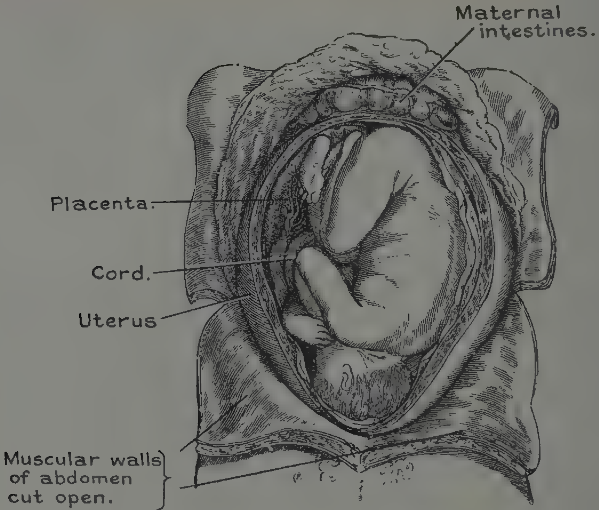

FIG. 67.—Attitude of the mature fœtus *in utero*.

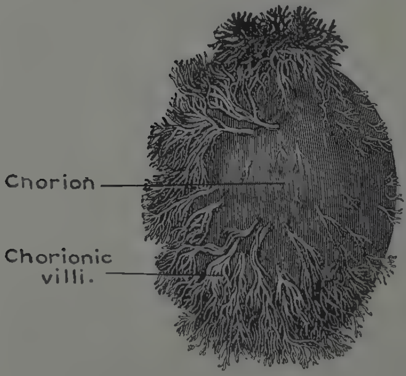

FIG. 68.—Human ovum of eighth week: the growth of villi preponderates at one part; at other parts the villi are already becoming atrophied. The embryo lies inside the sac. (After Carpenter.)

interchange takes place through the placenta or "after-birth," which is firmly attached to the uterus or womb on one side and to the other side of which is attached the cord. The placenta

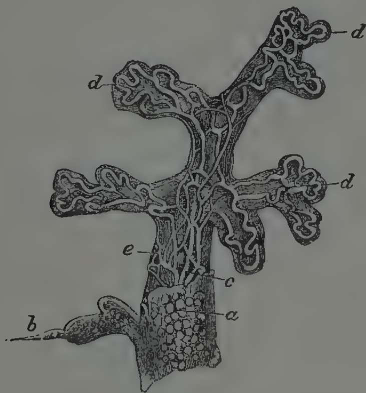

FIG. 69.—Chorionic villus magnified. *a*, epithelial covering; *b*, band uniting it to another villus; *c*, main arterial trunk of villus; *d*, terminal vascular loops; *e*, plexus of vessels between artery and vein. (After Ecker) × 350.

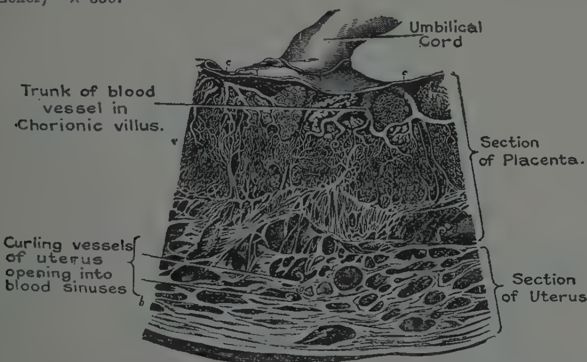

FIG. 70.—Section of fully-formed placenta, with part of the uterus. (After Galabin.)

acts as a sort of living filter, which allows useful material in the maternal blood to pass to the fœtus, but will generally—though by no means always—arrest any harmful substances which it may contain (see p. 7).

Structure of the Placenta.—The placenta consists of a very large number of finger-like processes called *chorionic villi*, which come from a portion of one of the membranes surrounding the embryo (figs. 68 and 69), and which are richly supplied with blood-vessels, interdigitating with corresponding hollows or vascular spaces, and called intervillous sinuses or spaces, in the wall of the uterus (fig. 70). In this way the blood (inside the villi) of the growing fœtus is brought into exceedingly close approximation with that of the mother (inside the intervillous sinuses). Now, although the two blood-streams are so close to each other, it is interesting to remember that never, throughout the whole period of pregnancy, is there any direct communication between the two bloods; the walls of the intravillous blood-vessels form a very thin but most effective barrier preventing the blood of one mixing with that of the other. That this is so is proved by a number of facts, viz. :—

(*a*) *Anatomical.*—On cutting microscopic sections of the placenta, one can actually see that, although the maternal blood empties into the uterine sinuses which receive the chorionic villi, the blood of the fœtus lies entirely inside the thin vessels accompanying these villi.

(*b*) *Experimental.*—Pregnant animals can be bled to death without at the same time causing loss of blood to the fœtus. Conversely, the animal fœtus, when still attached to its mother, can be bled to death without simultaneously causing loss of blood to the mother. Also, solid insoluble particles injected into the circulation of the one cannot be made to cross into the circulation of the other.

(*c*) *Chemico-Physical.*—If there were actual mixing of the two streams we should expect not only the microscopic picture but also the chemical composition and physical characters of the two bloods to be identical, but this is not the case.

(i) *Microscopically*, the numbers, both absolute and relative, as well as the appearances of the various corpuscles, are different in the two bloods. Thus, not only is there a larger number of red corpuscles per cubic millimetre in the fœtal than in the maternal blood, but nucleated red corpuscles, which are never found in normal healthy maternal blood, are normally found in the blood of the fœtus. Moreover, in certain blood diseases of the mother, where the maternal corpuscles are abnormal, either qualitatively or quantitatively, those of the fœtus remain normal.

(ii) *Chemically*, the various ingredients, such as hæmoglobin, iron, sodium, potassium, and other substances are present in different proportions in the two streams (see Table XCII, p. 256).

(iii) *Physically*, it has been found that certain of the physical constants, such as viscosity, fragility of the red cells, electrical conductivity, osmotic pressure (as determined by the depression in freezing point), &c., are different in the two fluids. In this connection it may be interesting to mention a striking fact showing the great strength of the delicate epithelial layer constituting the barrier between the two blood-streams. According to certain authorities, the depression in freezing point of fœtal blood is 0·579° C. below zero, whilst that of the maternal blood is 0·551° C. In other words, the freezing point of fœtal blood is 0·028° (=0·579−0·551°) lower than that of maternal blood. Now experiment shows that a difference of 1° C. in the freezing points of two solutions corresponds to a difference in osmotic pressure of twelve atmospheres between the two fluids; therefore the difference of 0·028° C. corresponds to a pressure of $0·028 \times 12 = 0·336$ atmospheres = 255 mm. of mercury, equivalent to the pressure of a column of water 3½ metres or 14 ft. high! Hence we come to the striking conclusion, if the data are correct, that the delicate epithelial layer between the two streams is strong enough to withstand such a very high pressure. It is, however, right to mention that other authorities have failed to find any difference in the freezing points of the two bloods.

Nutritive Functions of the Placenta.—Mixing of the two blood-streams being, therefore, ruled out, the question at once arises, "How does interchange of material occur between mother and fœtus?" That such interchange does occur is, of course obvious from the fact that the fœtus grows. The answer is that the various food materials required by the fœtus, as well as the various waste products excreted by it, pass from one circulation into the other by a kind of filtration. This, however, does not necessarily mean that the process is one of *physical diffusion* or *osmosis*, because if that were so, not only would all the substances in solution pass from one circulation into the other, but the various constituents would pass in proportion to their concentration (i.e., the percentage in which they were present in the transmitting stream). This, however is not the case, as

is shown by the following table, which represents the composition of specimens of blood obtained simultaneously from the mother and from the umbilical cord of the unsevered new-born infant.

TABLE XCII.

Nature of material	Maternal blood	Fœtal blood	Remarks
	mgm. per 100 c.c.		
Amino-acid nitrogen ..	5·9	7·9	Difference = 33 per cent. in favour of fœtus. Against osmosis
Sugar	132·0	115·0	Difference = 13 per cent. only in favour of mother. Probably osmosis
Fats	945·0	715·0	The differences in favour of the fœtus are so great as to preclude any possibility of passage by diffusion
Cholesterol	260·0	170·0	
Lecithin	280·0	200·0	
Total fats and lipoids ..	1,485·0	1,085	
Non-protein nitrogen ..	25·2	24·9	Almost exact balance. Osmosis almost certain
Urea nitrogen	10·5	10·4	Practically exact balance, indicating passage by osmosis
Creatinine	1·70	1·73	
Uric acid..	4·80	4·50	
Total excretory nitrogen from fœtus	17·00	16·63	
Mineral substances ..	713	741	The differences, all in favour of fœtus, are too great for diffusion by osmosis
K_2O	40	53	
Na_2O	353	385	
Immune bodies	Probably equal concentration		Probably osmosis

From this table we learn that whilst in the case of some substances the passage from one circulation to the other occurs by a process of physical osmosis, this is not so in the case of others, since their concentrations are totally different in the two streams. In the case of these latter substances transference occurs as the result of some *vital selective action* on the part of the chorionic epithelium allowing the constituents to cross from the mother to the fœtus, not in amounts that the mother can spare, but in quantities required by the fœtus, whether the mother can part with them or not.

The question of *physical osmosis versus vital selective action*

is not one of purely academic interest, but has a considerable practical importance from the point of view of ante-natal hygiene. As an illustration we shall take the question of contracted pelvis. If the substances pass through by a process of osmosis, then it should be possible in such cases so to regulate the mother's diet that, by diminishing the mineral concentration of her blood, the size of the fœtus and the softness of its bones could be so adjusted as to make it capable of passing through the maternal passages at full term without resorting to Cæsarean section or induction of premature labour with all their attending risks to both mother and fœtus—a possibility contended by Prochownik. The subject is one upon which a vast amount of research is still necessary. Blood analyses are liable to lead to erroneous conclusions, unless the water contents of the two bloods are known and taken into account. For this reason different observers have found different results. Table XCII, however, represents the results of the most reliable authorities. These results are confirmed by the experiences of the Great War. Notwithstanding the fact that many German expectant mothers were suffering from scurvy, osteo-malacia, &c., as the result of lack of mineral substances in their food, the number of infants born with similar conditions was small, suggesting that minerals pass from the mother to the fœtus by the selective action of the chorionic epithelium, and that therefore Prochownik's diet for reducing the size of the fœtus has no scientific basis.

Origin of Fœtal Fat.—The mode of transmission of fat is of considerable academic interest. The results of blood analyses, as well as physical considerations, definitely show that there can be no question of transference by diffusion. As to how the fœtus obtains its fat is not definitely settled. According to Hofbauer, the fat in the maternal blood is split up by placental enzymes into glycerine and fatty acid, in a manner similar to the splitting of fat in intestinal digestion and absorption, and that the separate constituents are re-synthesised inside the epithelial cells of the chorionic villi. Hofbauer found that when pregnant animals were fed with fat stained with Sudan red, which has an affinity for fat but not for fatty acids, stained fat can be seen in the intervillous spaces, but in the substance of the villi only unstained fat is to be seen. Other people, however, deny that fœtal fat is formed in that way. Thiemich, for instance, found that fœtal fat always has the same composition, no matter what the composition of the fat ingested by the

mother may have been. He believes, therefore, that fœtal fat is probably formed out of maternal glucose. If this is true, it is an indication for extra ingestion of carbohydrates by the mother during pregnancy. Slemms and his co-workers believe that the human fœtus probably makes its fat out of glucose, and attributes the excessive amount of fat in the blood of pregnant women to a preparation for lactation.

Rate of Transport of Material.—The fœtus grows most rapidly during the early months of pregnancy, although certain mineral substances are deposited at the highest rate during the last three months. This is seen from the following tables:—

TABLE XCIII.

Fœtal age ..	Water	Protein	Fat	Salts
6 weeks	97·5	Trace	Trace	Trace
4 months .. :	91·8	4·9	0·6	1
5 ,, ...	90·7	5·9	0·5	1·4
6 ,, ...	89·2	6·7	0·7	1·9
7 ,, ...	82·6	11·8	3·5	2 9
8 ,, ...	82·9	10·4	2.4	2·8
At term	74·7	12·6	8·7	3·3

TABLE XCIIIA.

	Weight in grm.	Dry weight in grm.	Nitrogen contents in grm.	Fat in grm.	Ash (salts) in grm.
Average weight of 7 months' fœtus	950	155	15	25	25
Average weight of full term fœtus	3,200	950	65	350	100
Addition in about 100 days ..	2,500	795	50	325	75
Average per day	22·5	7·95	0·5	3·25	0·75

From these tables we learn what are the **amounts of protein, fats and salts, daily required by the fœtus.**

Protein.—Table XCIIIA shows that during the last three months there is an average daily deposition of 0·5 grm. of nitrogen in the fœtal tissues. As nitrogen constitutes approximately one-sixth of the weight of protein, 0·5 grm. of nitrogen corresponds to about 3 grm. of protein. According to other observers, the daily deposition of protein is 6 grm. a day. In any case we can say that the fœtus requires about 3 to 6 grm. of protein a day. As the amount of protein taken by the mother

is usually more than 100 grm. a day, and is considerably in excess of her own physiological requirements, we see that the belief held by some to the effect that the mother requires much more protein food during pregnancy to provide for the needs of the fœtus is not founded on any scientific basis, and is indeed utterly erroneous and mischievous. There is little doubt that the excessive strain upon the kidney functions resulting from ingestion of excessive amounts of protein food by the mother is a potent factor in the causation of pregnancy toxæmia.

It is a fact that during the war, when food was scarce in Germany, there were fewer and less severe cases of eclampsia in that country. On the other hand, it has been suggested by those who believe that eclampsia may be due to some disturbance of calcium metabolism, that the diminution of eclampsia in Germany during the war was due to the greater consumption of vegetables containing calcium rather than to decreased consumption of protein.

E. D. Plass and L. Jean Rogert[1] examined the calcium and magnesium content of 205 cases of maternal blood during normal pregnancy, and compared the results with cases of pregnancy toxæmia, to test the theory that eclampsia is due to deprivation of calcium.

They find that although there is a definite diminished percentage of each of these salts in pregnant as compared with non-pregnant women, this is due not to an actual diminution in the amount of these substances but to the greater dilution of the blood (hydræmia or hydroplasmia). They further find that the amount of these substances is the same in the non-toxæmic as in the toxæmic women.

De Wesselow and Wyatt have found that during the pre eclamptic stage (i.e., in cases of slight œdema, albuminuria or raised blood-pressure) the amount of urea nitrogen in the blood is very little, if at all, increased, and therefore argue that it is not necessary to restrict protein in the majority of such cases. Indeed, they advocate a diet of milk, bread, butter, rice, eggs and fruit—*with a total restriction of salt.* Harding and Van Wyck[2] have treated ten such patients on high protein diet with restriction of salt and obtained good results. The matter, however, requires further investigation.

The mother requires very little, if any, nitrogen during the last few weeks of pregnancy, since a great deal of nitrogen has been retained by her during the early months. Of this retained nitrogen one-third goes to the fœtus.

Slemons gives the following Table XCIV of nitrogen metabolism during the later months of pregnancy:—

[1] *Amer. Journ. Obst. and Gynec.*, vi, 1923, p. 427.
[2] *Journ. Obst. and Gynæc. Brit. Emp.*, xxxiii, 1926, p. 17.

TABLE XCIV.

Type	Fluid taken	Amount of urine	Nitrogen in food	Nitrogen in urine	Nitrogen in fæces	Nitrogen balance
Primigravida ..	1,780 c.c.	1,306 c.c.	13·80 grm.	12 43 grm.	0·95 grm.	+ 0·42
Multigravida ..	1,890 ,,	1,007 ,,	16 77 ,,	13·26 ,,	0·53 ,,	+ 2·98
Twin pregnancy	1,135 ,,	1,135 ,,	15·00 ,,	8·28 ,,	2·00 ,,	+ 4·72

Hoffström found a storage of 310 grm. of nitrogen during the last twenty-four weeks of pregnancy. Of this about 100 grm. were needed for the fœtus; therefore 210 grm. must have been added to the maternal organism.

Similar experiments show also retention of mineral salts, viz., calcium, phosphorus, &c.

As for the fœtus, experiments have shown that practically the whole energy of its growth during the later months is derived from the metabolism of carbohydrates. Hence, it would seem that on physiological grounds the mother's diet towards the end of pregnancy should consist mainly of carbohydrates.

The experiments on which this conclusion is based consisted of a study of the proportion of the amount of carbon dioxide exhaled by the pregnant animal to that of oxygen inhaled by her during a prescribed period. This proportion $\frac{\text{volume of } CO_2}{\text{volume of } O_2}$ is called the *respiratory quotient*, and it is known that when the respiratory quotient is equal to unity then the substance being burned up in the tissues is carbohydrate, because this substance on combustion uses up one molecule of oxygen and produces one molecule of CO_2; (thus, $C_6H_{12}O_6 + 6O_2 = 6CO_2 + 6H_2O$, so that $\frac{CO_2}{O_2} = 1$); and when it is less than 1 it is either protein or fat, or a mixture in various proportions of protein, fat and carbohydrates, that is being metabolised. The amounts of oxygen inhaled and of CO_2 exhaled by a pregnant animal were determined before and during compression of the umbilical cord. The former give the combined metabolism of mother and fœtus, and the latter give that of the mother alone. The differences expressed as a ratio represent the respiratory quotient of the fœtus and this has been found to be equal to unity.

Influence of Excess of Protein in Maternal Diet upon the Fœtus.—Gladys Annie Hartwell[1] experimenting on rats, found that when the mother rat is fed on excessive amount of protein during gestation none of the young survive, even if the diet contains all the essential constituents.

[1] *Biochemical Journal*, xv, 1922.

Fats.—The same table, XCIIIA, shows a daily deposition of fat by the fœtus of about 3·25 grm. The practical conclusion derived from the fact is similar to that mentioned under protein. Indeed, it is questionable if the fœtal fat is derived from the maternal fat at all (see p. 257).

Salts.—The amount of salt fixed by the fœtus during the last three months of pregnancy is about two to three times that fixed during the whole of the first six months. In other words, the rate of mineral fixation is about four times as rapid during the last three months as during the first six months of pregnancy. This is especially the case in regard to calcium and iron. The daily amount of salt fixed by the fœtus is about $\frac{1}{6}$ grm. during the first six months and $\frac{2}{3}$ grm. per day during the last three months. This teaches us that during the last few weeks of pregnancy a diet rich in these mineral substances is indicated, e.g., brown bread, milk, cheese, eggs, spinach, green vegetables, &c., or there may be withdrawal of calcium and iron from the mother's bones or from her teeth. Decay of teeth, due to absorption of dental calcium by the fœtus, is not at all an uncommon complication of pregnancy; this is not only a painful condition, but is also a possible cause of pregnancy toxæmia, the result of absorption of toxins from the carious teeth. An interesting point in connection with absorption of iron is that infants born prematurely are anæmic to a degree corresponding with the extent of prematurity. The kidneys of a pregnant woman cannot deal easily with common table salt, and hence such salt is to be taken in as small quantities as possible.

An interesting case was recorded by Ballantyne of a woman who continually miscarried after the seventh month. None of the ordinary causes of miscarriage could be discovered in her case, and Ballantyne concluded that the cause probably lay in some derangement of the calcium metabolism of the mother. Administration of calcium to the mother in the last months of the next pregnancy resulted in the birth of a living infant at full term. The author has since had a somewhat similar case in his own practice. It is difficult to see why, if the experimental results are true, viz., that salts are transferred to the fœtus by vital action rather than osmosis, administration of calcium to the mother should have such an effect, but it is possible to explain it on the supposition that the extra calcium influenced the parathyroid metabolism of the mother and thus helped her to continue a pregnancy which would otherwise have terminated prematurely.

Storage of Iron and Oxygen in the Fœtal Tissues.—During the later periods of intra-uterine life there is a great storage of iron by the fœtus. This reserve of iron compensates for the deficiency of that substance in milk, which constitutes almost the sole article of diet of the infant during the greater part of the first year of post-natal life. There is also a storage of oxygen in the fœtal tissues, so that infants at birth can do without atmospheric oxygen for as long as twenty minutes without serious consequences. This latter fact, coupled with Eardley Holland's findings to the effect that obstetric traction of the head by a vigorous use of forceps, especially in the case of the after-coming head in breech presentation, is liable to lacerate the fœtal brain, teaches us that not only is it a mistake to expedite unduly the birth of an after-coming head on the grounds that prolonged compression of the cord might suffocate the fœtus, but it also follows that such breech presentations should be converted into vertex presentations before labour, and the use of forceps should, as far as possible, be avoided altogether by proper ante-natal supervision and treatment. In this connection the author would earnestly advocate confirmation of the position of the fœtus, towards the end of pregnancy, by means of the X-rays, as breech presentations have been mistaken for vertex by the most expert obstetricians (see Plate opposite).

Transmission of Waste Products from Fœtus to Mother.—That substances can and do pass in the opposite direction, i.e., from fœtus to mother, has been demonstrated experimentally by injecting strychnine and hydrocyanic acid into an animal fœtus while still in the uterus, when the mother died from strychnine or prussic acid poisoning. Blood analyses of excretory nitrogen in fœtus and mother (see p. 256) show that the products of fœtal excretion are passed on to the maternal circulation by a process of osmosis. Whilst normally oxygen passes from mother to fœtus, it was shown by Zweifel that in animals, during a state of asphyxia of the mother, oxygen passes back from the fœtus to the mother; further, that when artificial respiration is done to the mother the oxygen goes back from the mother to the fœtus. These facts teach us that the home of an expectant mother should be well ventilated, not only for the sake of the mother but for that of her fœtus. It also explains why women with heart disease, who suffer from lack of oxygenation of their blood, often have premature labours with dead fœtuses. In addition we learn from this fact that whilst immediate post-mortem Cæsarean section on a woman accidentally killed will probably

Skiagram of fœtus inside the uterus during 32nd week of pregnancy, showing vertex presentation. The clinical diagnosis was breech presentation (Dr. Ulysses Williams).

result in the birth of a living infant, and indeed cases have been recorded of living infants having been thus extracted twenty-three minutes after their mother's death, such operation is, as a rule, useless in cases of maternal death from prolonged illness, since the fœtus probably succumbed to asphyxia before the death of its mother.

It has also been shown that various *internal secretions* may pass through the placenta in either direction as occasion requires.

Passage of Poisons, Toxins and Microbes.—(*a*) *Poisons.*—The poisons that have been specially investigated are alcohol and lead.

(I) *Alcohol.*—Numerous experiments upon animals as well as women have shown that alcohol given to the pregnant mother passes into the fœtal circulation and is found in its tissues. In the case of women, for instance, Nicloux found that when mothers were given a strong dose of alcohol, about an hour before delivery, analysis of the blood taken from the umbilical cord of the infant immediately after birth showed the presence of alcohol. Recent work by Madame Dr. Smilya A. Kostitch,[1] of Belgrade, on pregnant sows fed with bread soaked in a solution of alcohol a few hours before they were killed and their fœtuses extracted, has shown that not only did the alcohol pass into the blood but also into the various organs of the fœtus, including the sexual organs. It also passed into the mother's milk. The following is an example of her findings:—

Sow weighing 80 kg., fed on bread soaked in 800 c.c. of a 10 per cent. solution of alcohol (i.e., 1 c.c. of pure alcohol per kg. body weight). Killed four hours and forty minutes after the feed. Analysis of fœtal tissues gave the following results:—(see Table XCV).

TABLE XCV.

	Weight	Amount of alcohol	Percentage of alcohol
Blood	6·30 grm.	0·0065	0·10
Liver	4·12 ,,	0·0041	0·10
Ovaries	6·88 ,,	0·0042	0·06
Brain	2·67 ,,	0·0022	0·08
Kidneys	4·95 ,,	0·0042	0·08
Muscles	4·08 ,,	0·0032	0·08
Liquor amnii	7·20 ,,	0·0066	0·09
Fœtal liver	4·21 ,,	0·043	0·10

[1] "Du passage de l'alcool ingéré dans les principaux tissus et organes." *Revue Internationale Contre l'alcoolisme*, 1922.

In the case of male fœtuses, alcohol could be detected in considerable quantities in the testes and vesiculæ seminales.

Experiment has also shown that within an hour and a half of ingestion there is established osmotic equilibrium for alcohol between the maternal and the fœtal bloods. As fœtal tissue is very delicate and in a state of rapid growth, it is inferred that alcohol in considerable quantities must have a deleterious effect not only upon the fœtus but upon the next generation, since the fœtal sexual glands are also injured by the poison.

(II) In addition to alcohol, it has been shown experimentally that such substances as lead, phosphorus, mercury, nicotine, morphine, chloroform, iodides, arsenobenzol preparations (salvarsan, &c.), and other poisons or drugs, pass from mother to fœtus (see p. 117). Hence, it is not only possible for the fœtus to be poisoned by toxins or poisons circulating in the maternal blood—a point to be remembered in connection with scopolamine-morphine anæsthesia, or twilight sleep—but certain fœtal diseases, especially syphilis, can be treated with certainty by administering the appropriate therapeutic agent to the mother. Ballantyne even recommends the administration of calcium salts to expectant mothers coming from a hæmophilic stock, with the idea of preventing hæmophilia in the unborn child. It has also been shown that immune bodies pass from mother to fœtus, and hence prematurely-born babies are more liable to infection because of their having been deprived of a considerable amount of such protective substances. It also follows that expectant mothers should be protected from dangerous industries such as lead, phosphorus, and perhaps tobacco factories, and such women should refrain from excessive indulgence in alcohol or tobacco.

(*b*) **Microbes.**—Apart from syphilis, the transmission of microbes through the placenta is extremely rare, but it does occur. The *Bacillus typhosus* has been found in fœtal blood and tissues in a number of cases, and infants have sometimes been born with evidence of small-pox—the most celebrated case of this nature having been Mauriceau, the famous accoucheur of the seventeenth century, who was born pock-marked, his mother having caught the disease in the later months of pregnancy. Indeed, a pregnant woman who had had small-pox or had been vaccinated may associate with cases of small-pox with impunity to herself but with danger of transmitting the disease to her unborn infant. A case has been recorded of an infant born dead from small-pox caught indirectly from its father through its

vaccinated mother, who had herself escaped infection. On the other hand, vaccination of the mother temporarily protects the fœtus.

A very remarkable case, which it is impossible to explain, was published by Chantreuil in 1870, of binovular twins born to a mother who had escaped small-pox. One of these showed signs of intra-uterine small-pox, and the other was free from the disease. Similar cases of twins, one of which was congenitally syphilitic and the other was healthy, have also been recorded.

Infants have also been born with the eruption of measles on them. Congenital tuberculosis, with tubercle bacilli in the fœtal tissues, is extraordinarily rare, no more than about twenty cases having been described in the literature. As a rule the infants born to tuberculous mothers are very healthy and may actually be above the average birth weight. Cholera, malaria and pneumonia in the fœtus have also been recorded.

We see, therefore, that the older views, e.g., of Davaine, 1865, and of Brauell, 1875, to the effect that the placenta forms an impassable barrier against microbic invasion of the fœtus is no more tenable. The passage is favoured by the presence of toxins in the circulation which render the placenta permeable.

Passage of Tuberculous Antitoxin through the Placenta.—Since patients with mild tuberculosis produce antibodies which help to cure the disease, it is possible that the fœtus of a mildly tuberculous woman may become temporarily protected against the disease by the transference of these antibodies to its own blood.

From all this it follows that pregnant women must be protected from exposure to infectious diseases.

J. R. Learmouth[1] has shown that:—

(1) Agglutinin for *B. typhosus* is found in the serum of the young of immunised female guinea-pigs.
(2) The agglutinin is present before injection of colostrum.
(3) Agglutinin as such may pass through the placenta.
(4) The titre of the fœtal may equal or exceed that of the maternal serum, but there is no definite quantitative relationship.
(5) The male cannot transmit acquired immunity.

The matings employed were:—

(1) Non-immunised males × non-immunised females.
(2) Immunised males × non-immunised females.
(3) Non-immunised males × immunised females.
(4) Immunised males × immunised females.

[1] *Journ. Hyg.*, xxii, 1923-24, p. 100.

Transmission of Gaseous Substances.—As early as 1674 John Mayow regarded the placenta as the fœtal lung, and Ray, in 1759, compared the chorionic villi to the gills of a fish. In 1798 Scheele noticed that the blood in the umbilical vein, carrying blood to the fœtus, was brighter in colour than that in the umbilical artery which carried back the fœtal blood to the mother. It was not, however, till 1876, when Zweifel demonstrated spectroscopically the presence of oxyhæmoglobin in the umbilical vein before the fœtus breathed through its lungs, that the transference of oxygen from the mother to the fœtus through the placenta was universally recognised. The same observer also showed that experimental asphyxia of the mother in animals made the oxygen pass in the opposite direction from the fœtus to the mother, and that artificial respiration performed on the mother restored the original direction of the oxygen from mother to fœtus. Cohnstein and Zunz, in 1884, showed that umbilical arterial blood, i.e., blood going from fœtus to mother, contains 4·67 per cent. less oxygen and 4·72 per cent. more CO_2 than umbilical venous blood, i.e., blood coming from mother to fœtus. Hence there can be no doubt that oxygen passes from mother to fœtus and carbon dioxide passes in the opposite direction. The CO_2 is almost certainly transmitted by a process of osmosis, but the oxygen is almost equally certainly taken up by the chorionic villi by some vital activity, since the oxygen is combined with hæmoglobin, which is a colloid that cannot diffuse through the villi. It is most probable that before the oxygen can be passed on to the fœtus it is first split off from the oxyhæmoglobin by the agency of some placental enzyme.

It may be pointed out also that the fact that the umbilical venous blood contains an excess of oxygen approximately equal to that of carbon dioxide in the umbilical arterial blood, shows that the respiratory quotient of the fœtus is approximately equal to unity, a fact the importance of which I have pointed out before (see p. 260).

Parasitism versus Symbiosis.—Although I have stated (p. 251) that the fœtus lives a sort of parasitic existence upon its mother, it must not be supposed that it derives all the good from her without giving her anything useful in return. There are a number of facts to show that the fœtus and its mother live rather in a state of *harmonious symbiosis* or partnership, each one contributing something useful to the other. We have seen

that in maternal asphyxia the fœtus will give to the mother some of its own oxygen, even at the cost of its own life. Further, careful experiments, in animals as well as on women, have shown that during pregnancy a larger amount of nourishment is derived by the mother out of the food that she takes and part of which she passes on to the fœtus. Indeed, it is a clinical fact that in many cases of anæmia the mother feels better during pregnancy than at any other time. Another interesting fact is that, in cases of need, internal secretions pass from the fœtus to the mother. Thus, while experimental removal of the pancreas in animals gives rise to diabetes, the same operation performed on pregnant animals is not followed by the appearance of sugar in the urine, because the insulin of the fœtal pancreas passes to the mother to replace her own missing insulin. The same does not, however, hold good in human diabetes, which, according to some, is generally made worse by pregnancy. (But see below, p. 281.)

Fœtal Diseases.—To Dr. J. W. Ballantyne belongs the great credit of having created the subject of ante-natal pathology.

(A) **Transmitted Diseases.**

The fœtus is liable to most of the infectious diseases that attack the mother, as well as to some of the chronic infections or other diseases which may be conveyed to it through the placental circulation. Syphilis in the majority of cases passes from mother to fœtus, tuberculosis sometimes, though very rarely, does so, but poisons and toxins, as well as other substances present in excess in the maternal blood, frequently pass into the fœtal circulation and affect the fœtus. Thus lead, opium, alcohol and other drugs, sugar from a diabetic mother, and toxins in cases of pregnancy toxæmia, are transferred from mother to fœtus. On the other hand, antitoxins and other protective substances also get transferred from mother to fœtus.

(B) **Diseases originating in the Fœtus.**

In addition to the above transmitted diseases, the fœtus has a special pathology of its own. It will not serve any useful purpose to enumerate the primary diseases of the fœtus in a book dealing with hygiene, since very few, if any, of these can, in the present state of our ignorance of their pathogenesis, be prevented. These diseases, however, may be divided into two main groups, viz.:—

(a) *Diseases which may attack the adult or the child after birth*:—

(1) Congenital fœtal dropsy, ascites, peritonitis, elephantiasis, &c.
(2) Skin diseases, e.g., ichthyosis, scleroma, &c.
(3) Bone diseases, e.g., achondroplasia.
(4) Gastro-intestinal diseases, e.g., congenital pyloric stenosis, stenosis of other parts of the tract, e.g., œsophageal, duodenal, intestinal, &c.

(5) Kidney diseases, e.g., congenital cystic kidneys.

(6) Heart diseases, e.g., endocarditis.

(*b*) *Diseases peculiar to the fœtus*, generally designated as congenital malformations or monstrosities. The study of these constitutes the subject of Teratology.

Causation of Monstrosities.—On the basis of the pioneer work of Dareste, Allen, Thomson, the brothers St. Hillaire, Bovers, Roux, Ballantyne, and others, we may classify the causes of fœtal monstrosities as follows:—

(A) **Germinal.**

(1) *Heredity.*—We know, for instance, that hare-lip and similar malformations are hereditary Mendelian characters.

(2) *Influence of physical or chemical agencies upon the reproductive cells of the parents.*

Bardeen produced monstrosities in the offspring of toads by exposing the parental sperm to the X-rays. The influence of alcohol has been discussed in considerable detail on p. 263.

(B) **Placental.**

(3) *Influence of toxins, &c., transmitted from the maternal circulation.*

Guyer and Smith produced rabbits with defective eyes by injecting the pregnant mother with the serum of fowls rendered immune by repeated injection of pulped rabbits' lenses. This deformity was transmitted by male and female to subsequent generations.

(4) Arrest of development the result of thermal, chemical or other physical agencies.

Thus Stockard and Lewis produced 50 per cent. of Cyclopean monsters from the eggs of the common minnow by treating them with solutions of magnesium chloride.

(5) *Amniotic adhesions.*

(C) **External.**

(6) *Injuries to abdomen or uterus.*

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CHAPTER IX

THE CARE OF THE EXPECTANT MOTHER

> "Don't, till 'tis Born, defer thy Pious Care,
> Begin betime, and for its Birth prepare."
> SCEVOLE DE ST. MARTHE (Sixteenth Century).

THE care of the expectant mother has a twofold object, viz.: (1) To protect the woman herself against any of the accidents that may be associated with pregnancy, labour and the puerperium; (2) to preserve the life and welfare of the child before and during birth, and to ensure, as far as possible, that it will be born strong enough to withstand the rigours of its new surroundings during the most critical period of its early post-natal (i.e., neo-natal) life.

From what I have said in previous chapters regarding the causes of maternal and fœtal mortality, as well as about the physiological connection between the mother and the fœtus, the following account of the hygiene and mode of life of the expectant mother will need very little explanation.

Scevole de St. Marthe, from whose "Pædotrophia" I have given the quotation at the heading of this chapter, gives the following rules to an expectant mother:—

> "Let neither Grief, nor Fear, nor boundless Joy,
> The Peace and Vigour of thy Mind destroy.
> . . . .
> Refresh thy weary Limbs with sweet Repose,
> And when fatigued thy heavy Eye-lids close.
> . . . .
> . . . be careful, how your Meats you chuse,
> And chosen well, with Moderation use.
> With too much Food your Stomach ne'er oppress
> And let it as 'tis richer, be the less.
> . . . .
> Mix Water with your Wine to quench your Thirst,
> And never let the last exceed the first.
> Fruit, Herbs, and Sallads when the Body's dry
> The want of Moisture will as well supply."

Diet.—The diet throughout pregnancy should be adequate but not excessive in amount, easily digestible and nutritious.

The expectant mother must not, as is frequently recommended by lay persons, "eat for two," since, as we have already seen (p. 258), her growing fœtus requires comparatively little over and above what the woman generally eats when not pregnant. Excess of food may not only result in indigestion and tend to make her abdominal muscles, which have an important function to perform in the expulsion of the fœtus during the second stage of labour, weak and flabby, but excess of protein food may also result in toxæmia. She must eat enough and satisfy her appetite and no more. Meat should be taken in small quantities and not more than once a day; and rich, indigestible, as well as highly seasoned articles of food should be avoided, in order to give the kidneys as little work to do as possible. The woman must take butter, fresh vegetables in the form of salads, lettuce, spinach, beans, peas, tomatoes, &c., and fresh fruit (prunes, figs, dates, &c.), as well as coarse brown or whole-meal bread. These will not only provide a good supply of mineral salts and of vitamins for herself and the fœtus, but will also help to keep her bowels regular. The mother's need for a sufficiency of mineral salts is especially important during the later months of pregnancy in order to provide for the need of the fœtus (see p. 261), otherwise the fœtus will take mineral substances from the mother's tissues. Thus, it will take some minerals from her teeth, causing her dental decay with all its unpleasantness and dangers. It may take some calcium from her bones, causing them to soften (osteomalacia). It may also take some iron from her blood by breaking up her red blood-corpuscles, giving rise not only to anæmia but also to jaundice, as the result of the breaking up of the hæmoglobin. She must drink a sufficient quantity of water between meals to ensure a proper flushing of the kidneys, and a glass of water at bed-time as well as the first thing in the morning often acts as an aperient. Between two and three pints of water should be drunk during the day. Alcohol should preferably be excluded altogether from a pregnant woman's dietary, and strong tea or coffee is to be taken sparingly. Smoking also is best done without.

The following is a sample dietary sheet for an expectant mother :—

Breakfast.—Oatmeal porridge with milk, bread (preferably brown) and butter, marmalade, an egg and a cup of cocoa.

Lunch.—Egg omelette, bread and butter, rice or tapioca pudding, a piece of cheese.

Tea.—Bread and butter, an egg, a glass of milk or a cup of cocoa.

Dinner.—Vegetable soup, 3 or 4 oz. of fish or chicken with two vegetables, bread and butter and stewed fruit.

Regularity of the Bowels and Prevention of Hæmorrhoids.—Constipation, which is often troublesome during pregnancy on account of the interference of the growing womb with the normal peristalsis of the intestines, must be avoided, since not only does constipation predispose to pregnancy toxæmia or give rise to hæmorrhoids which are very unpleasant, but a full rectum during labour may be the cause of puerperal sepsis. A daily action of the bowels may, in many cases, be ensured by a proper diet containing a sufficient amount of vegetables and fruit of the kinds mentioned in the last paragraph, brown bread, and a reasonable amount of water taken during the day. The woman must also cultivate a habit of going to the closet at a regular and fixed time of the day. General and special exercises (see below) are also very helpful for the purpose. If drugs are necessary, mild aperients such as about fifteen drops of liquid extract of cascara sagrada, or a tablespoonful of liquid paraffin at bed-time, are least harmful. But it is best not to resort to drugs without first consulting a doctor.

Dress.—The clothing should be loose and so arranged as not to cause undue pressure upon the uterus, breasts and other parts of the body, such as the veins of the legs which are liable to become varicose. All the clothes should, therefore, as far as possible, be suspended from the shoulders, and no corsets, tight waist-bands or garters may be worn. The French term for pregnancy is *enceinte*, which is derived from the Latin *incincta*, meaning ungirdled, because it was the custom among the Romans to dispense with the girdle as soon as pregnancy set in. "No expectant mother," says Ballantyne, "can hope to be comfortable and in the height of fashion at the same time, for those who rule in the autocratic realms of ladies' dress do not legislate for pregnancies." Multiparous women with pendulous abdomens should wear properly fitting, but not too tight, belts. The stockings should be warm, and the boots or shoes must be easy-fitting, fairly thick-soled to protect against damp, and low-heeled. The large abdomen, by throwing the centre of gravity of the body forward, causes the head and upper part of the spine to be thrown backwards in order to balance the body, and high heels necessarily throw the abdomen still further forward, thus entailing an extra strain on the muscles of the head, neck, and thoracic spine, as well as undue stretching of the abdomen.

Exercise and Rest.—Amongst the Spartans young women had to do various exercises to strengthen the abdominal muscles, so

that when they became pregnant they should "more easily do away with the pains of child-bearing." In addition to her ordinary domestic activities, a pregnant woman must take a sufficient amount of outdoor exercise, such as regular daily walks. In addition, it is recommended that she practise every morning and evening for about five or ten minutes at a time a few simple exercises specially designed to strengthen her abdominal muscles (fig. 79). These will not only help her to have an easy labour, but are useful in keeping her bowels regular. She is to lie flat on her back and raise first one leg, then the other.

FIG. 71.—(From Ashton's "Gynæcology," published by W. B. Saunders & Co.)

Fixing her feet under some heavy object she should then raise her body into the sitting posture. If the woman has had several pregnancies without mishap she may indulge in some more strenuous exercise, but during a first pregnancy the exercise should be of the gentlest nature. Working women who are employed in factories are more in need of rest than exercise during the later months of pregnancy. Sea voyages are not to be undertaken during first pregnancies or during any other pregnancy if the woman has had a previous miscarriage.

Sleep.—A pregnant woman is in need of plenty of sleep, at least nine hours a night and a short nap in the afternoon.

Bathing.—Baths, neither too hot nor too cold, should be taken at least twice a week before going to bed, so as to counterbalance

any possible fatigue and to promote sound unbroken sleep. The baths also help the elimination of waste material through the skin. A bath temperature of about 98° F. is the proper temperature. Sea-bathing as well as Turkish baths should be avoided, at any rate during a first pregnancy when the tendency to abortion is unknown, and by women who are not accustomed to it, for fear of bringing on a miscarriage.

Care of the Breasts.—Towards the end of pregnancy secretion often oozes out of the nipples. This may dry and leave scales which adhere and expose a raw surface when detached, with consequent danger of mammary infection and abscess. The nipples should therefore be gently brushed daily with a soft toothbrush and soap and water. If the nipples are small and flattened they should be gently drawn out night and morning to render them serviceable to the infant.

Care of Teeth, &c.—All discoverable sources of sepsis about the mother must be removed, and the teeth especially must be kept very clean and in a healthy condition. Dental caries or pyorrhœa must be put right, as, apart from the unpleasantness of these conditions, all septic foci are possible causes of pregnancy toxæmia or puerperal sepsis.

Dental Caries as a Cause of Pregnancy Toxæmia.

That dental sepsis can cause pathological conditions in the body has, of course, been emphasised by numerous observers. Rosenow and Meisser proved the relationship experimentally by removing the pulp from the lower bicuspids of a number of animals, and after introducing suspensions of bacteria into the pulp chambers, the canals were properly covered and sealed. Five out of six dogs infected with streptococci from the urine of patients with renal calculus developed stones from which the streptococci could be isolated, and when these latter were injected intravenously into rabbits renal stones were formed in them. Control animals showed no such lesions.

Talbot has shown a definite relation between foci of infection in teeth and placental infarction.

Mental Hygiene.—All pregnant women must, as far as possible, be protected from shocks, worries and anxieties, since violent emotions undoubtedly tend to produce miscarriage. It has been proved experimentally in animals that sudden frights will cause an increased production of adrenalin, which, as we know, has a stimulating effect upon the uterus, causing it to contract and expel the fœtus.

Maternal Impressions.—The belief that any impression received by the expectant mother is, as it were, photographed upon her fœtus so that the latter is born with some structural

alteration closely simulating such an impression, dates from the times of remotest antiquity (see p. 21), and is of practically world-wide distribution. A baby, for instance, is born with a certain deformity or a birth-mark, and the mother often attributes the condition to the fact that during some part of her pregnancy she either saw a person similarly afflicted or that she touched a corresponding part of her own body whilst shocked by the sight of some horrible object or a fire.

The belief was pushed to an absurd length when Bartholin made the statement that a lady conceived a child by her husband who had been absent from her for some years through the influence of her imagination alone! Cases of alleged maternal impression are every now and again recorded in the lay as well as in the medical press, but as far as our present knowledge goes, not only is there apparently no scientific foundation for the belief, but there is very good reason against it. Ballantyne classifies the **arguments against this belief in maternal impressions** as follows :—

(1) *Experiential.*—(*a*) Nearly every pregnant woman sees many cases of deformity or other unpleasant sights during her pregnancy, and yet the number of infants born with reproductions of such sights on their bodies is exceedingly small. (*b*) Many cases of congenital deformities or birth-marks occur where no previous maternal impressions can be traced.

(2) *Logic.*—Since a woman cannot by her own impressions mark her own body, it is not likely that she would be able to mark her unborn infant's body in that way.

(3) *Scientific Considerations.*—(*a*) *Anatomical :* (i) There is no nervous or *direct* circulatory communication between mother and fœtus (see p. 254). (ii) Monstrosities observed in human fœtuses are also found in animals. (*b*) *Embryological :* By the end of the sixth week of gestation nearly all the fœtal external and internal conformation is complete (see fig. 4). In other words, the main parts of the fœtal body are completely shaped before the mother is quite aware of her pregnancy, and therefore no impression received after that time can have an influence upon the fœtal bodily conformation.

But whilst there seems to be no scientific basis for the antenatal psycho-photographic reproduction of maternal impressions upon corresponding portions of the fœtal body, it is possible to associate, on theoretical grounds, certain deformities in the fœtus with some violent mental shock received by the mother during pregnancy.

We have seen that shock will call forth an excessive production of adrenalin. The stimulating action of this hormone upon the uterus may cause detachment of the placenta to a varying extent. If the detachment is large, the fœtus will die and be expelled. If it is small, a part of the fœtus may be deprived of its normal blood-supply and fail to grow properly. Moreover, the various hormones produced by the emotion of the mother may pass through the placenta to the embryo or fœtus. These may act upon the latter in some selective chemical manner upon certain parts of its developing or growing body, or they may excite the fœtus to excessive movements which bring about coiling of the cord round a limb, resulting in spontaneous amputations, &c. For all these reasons it is necessary to protect the expectant mother, not only against all sorts of injury but also against any emotion or unpleasant sights or news.

Claude Quillet, physician and poet of the seventeenth century, thus speaks of maternal impressions:—

> "As when the Wheaten Mass is worked to Dough,
> Or swells with Leaven in the Kneeding Trough,
> It takes Whatever marks the Maker gives
> And from the Baker's Hand its Form receives.
> So works the Fancy on the Female Mould
> And Women should beware What they behold.
> . . . .
> . . . .
> Since by foul Objects filthy Births are made
> And the vile Picture's to the Womb convey'd,
> A pregnant Wife will ne'er behold a Whale
> Nor Porpus nor the Dolphin's Azure Scale.
> Nor thee, Oh Proteus, will she see, nor you
> Tritonian Monsters, While she's Teeming, view.
> But let her on the lovely Nereids gaze
> And fix her Eyes on ev'ry Charming Face."

Sexual Hygiene.—To avoid risk of abortion, women, especially in a first pregnancy, should abstain from sexual intercourse during the first three months, especially at times when the periods would have been due if pregnancy had not been present. The third month is the one during which abortion occurs with the greatest frequency, and therefore during that month the rule with regard to abstinence should be particularly observed. Coitus during the last few weeks of pregnancy must be particularly avoided, because of the risk of puerperal septicæmia resulting from dangerous organisms introduced into the woman's genital tract in that way (see p. 198).

Housing.—The expectant mother must live in a comfortable, sunny and well-ventilated house. Her bedroom should be of good size and should have a good supply of fresh air. Imperfect ventilation interfering with proper aeration of blood must, as we have seen (p. 262), have a bad effect upon the fœtus.

Occupation.—Statistics have been published by various observers to show that if employed women cease work during the last few weeks of pregnancy, the stillbirth-rate, as well as the neo-natal mortality amongst their infants is much lower than among infants whose mothers worked right up to the end of pregnancy. The following figures, published by the United States Children's Bureau, are based upon 24,000 observations, and are given by Dr. Josephine Baker in her "Child Hygiene":—

TABLE XCVI.

Employment of mother	Stillbirth-rate per cent.	Neo-natal mortality rate per 1,000	Premature births rate per cent.
(1) Employed away from home	4·9	63·2	6·1
(2) Employed at home (kept lodgers, &c.)	3·5	36·7	3·5
(3) Mother employed (did house work..	3·1	43·1	5·2

These figures show that employment of mothers away from home entails higher prematurity, stillbirth and neo-natal mortality rates amongst their infants. The higher rates amongst mothers not employed at all, as compared with those among mothers employed at home, may be due to the fact that the group of mothers who were not employed included mothers who were suffering from ill-health. On the other hand, without further particulars it is impossible to draw any safe conclusion regarding the influence of maternal work away from home upon the fœtus, since it is possible that the mothers included in this group may have had a considerably higher percentage of syphilitic women amongst them than those in the other two groups. Dr. Ramsay, of America, during his war work in France, found that when women worked in factories up to the time of their confinement the neo-natal mortality amongst their infants was sometimes as high as 50 per cent., whilst amongst women who worked on farms the neo-natal mortality of the infants was only 8·5 per cent., although the work was quite as hard as in the factories. He further found that when

the factory women were relieved of their work a month before confinement, and proper food and rest given them, the neo-natal mortality amongst their infants was reduced to a minimum, because the mothers were able to suckle their babies. The only safe conclusion that can be drawn from these statistics is not that maternal work *per se* is prejudicial to the welfare of the fœtus but that occupation involving prolonged confinement in badly ventilated factories is harmful, because the fœtus must part with some of its oxygen to the mother, and the ill-health of the mother renders her subsequently unable to suckle the infant. There is no doubt, however, that certain trades, such as those involving the handling of lead, mercury, phosphorus, &c., are dangerous, not only for the woman but also for her fœtus.

Miss Elderton[1] finds that the correlation coefficient between heavy work of mother during pregnancy and difficult labour is *nil*. The same is the case for maternal health and difficulty of labour. The coefficient of correlation between first pregnancy and difficult labour is, however, considerable, viz., $0{\cdot}279 \pm 0{\cdot}042$.

Protection against Infectious Diseases.—From what has been said on p. 264, it is clear that a pregnant woman must be protected from exposure to infectious diseases, both for her own sake and for the sake of her fœtus. Also in the case of a small-pox epidemic, she must be vaccinated or revaccinated.

Prevention of Abortion.—Abortion is most likely to happen at times which would have been menstrual periods if pregnancy had not occurred, because at those times the uterine congestion, as well as the woman's nervous excitability, is greatest. Hence special precautions should be taken by expectant mothers at those dates, especially during a first pregnancy, or if they are known to be susceptible to abortion. Abortion is also most likely to occur during the first four months, but especially during the third month of pregnancy, and hence particular care is necessary during that time. There are many causes of abortion which are not preventable, but the risk can be minimised by avoiding nervous shock, such as fright, anxiety, sight of startling scenes, excessive suckling, or sexual indulgence, hot or cold baths in women unaccustomed to them, violent purgation, vaginal douching, &c.

Syphilis as a cause of abortion is dealt with on p. 108.

[1] Ethel M. Elderton, "On the Relative Value of Factors which Influence Infant Mortality," *Annals of Eugenics*, 1925, vol. i, p. 231.

Voluntarily induced abortion is a criminal offence, and if the woman loses her life as the result of such action the person responsible is liable to capital punishment.

Medical Supervision.—Throughout the whole of pregnancy every woman must be under proper medical supervision. It is quite certain that efficient ante-natal supervision would result in a 50 per cent. reduction of maternal and fœtal as well as neo-natal mortality. In addition, it is safe to say that such medical supervision, by obviating instrumental and manual manipulations during a difficult labour, would also prevent a large part of chronic invalidism of mothers. The doctor's supervision consists of the following :—

(1) **Diagnosis of pregnancy,** and if possible to ascertain the **date on which the confinement is to take place,** so that the woman could make the necessary preparations.

(2) **Examination with regard to the woman's fitness for the ordeal of pregnancy and labour**, although the best time for such examination is before the occurrence of pregnancy. In the course of such examination the medical attendant examines the heart and circulatory system, as well as the lungs, since advanced disease of these organs might necessitate induction of abortion. He will also detect syphilis, &c., by the Wassermann reaction if necessary, and treat it.

The following figures by H. Boas and S. A. Gammeltoft[1] show the **relative values of mercury and salvarsan in the treatment of ante-natal syphilis.** Out of 111 expectant mothers treated with mercury 31 gave birth to normal living babies, i.e., a proportion of 28 per cent., whilst out of 79 such women treated with salvarsan the number of normal babies born alive was 60, i.e., 75 per cent.

In addition to the figures already stated in Chapter IV, p. 109, regarding the effect of ante-natal treatment of syphilis, the following may be given: John Adams[2] had 8 stillbirths amongst 113 treated mothers (i.e., 7 per cent.). W. E. Welz and A. E. Van Nest[3] had a fœtal mortality of 2·2 per cent. (i.e., 1 out of 46) amongst mothers adequately treated, and one of 26 per cent. (26 out of 100) amongst those insufficiently treated. Vignes and Galliott[4] give the following table :—

[1] *Act. Gynæcol. Scand.*, i, 1922.

[2] *Brit. Med. Journ.*, 1922.

[3] *Amer. Journ. Obstet. and Gyn.*, August, 1922.

[4] *Progrès Méd.*, 38, 1923.

Treatment	No. of Mothers	Living Children	Dead Children	Percentage of Stillbirths
Before and during pregnancy	21	21	0	0
During pregnancy only ..	29	27	2	7
Partial, during pregnancy ..	16	9	7	44
No treatment	53	20	33	62

(3) **Watching for signs of pregnancy toxæmia.** A pregnant woman, especially during a first pregnancy, may, for various reasons into which it is not necessary to enter in detail here, suffer from the circulation of toxins or poisons in her blood. These poisons may come either from the fœtus or from some deranged metabolism in the mother, and, on account of the excessive strain thrown upon her kidneys which have to eliminate both her own as well as the fœtal waste products, may not easily be excreted in her urine. As a result of this the poisons are retained in the mother's blood. The predisposition to such a condition is aided by faulty diet (see p 259), the presence of septic foci such as carious teeth (p 273), septic tonsils, &c., as well as by chronic constipation, allowing absorption of toxins to take place from the intestines.

Pregnancy toxæmia may manifest itself in a variety of ways, of which the most important ones are eclampsia or convulsions, and the so-called pernicious vomiting, or hyperemesis gravidarum.

The danger signals which the doctor will look for are :—

(*a*) *Those associated with the nervous system*, such as headache, dizziness, hyperexcitability, or melancholia due to the action of the toxin on the brain, and dimness of vision due to albuminuric retinitis (see "Examination of Urine" below).

(*b*) *The skin*, such as œdema of the ankles or eyelids.

(*c*) *Circulatory system*, such as raised blood-pressure (anything over 130 mm.).

(*d*) *Urinary system.*—Attention is paid to the quantity of urine passed daily. Any diminution in the daily amount (i.e. less than fifty ounces), especially if it is progressive, is a very suspicious sign of the onset of toxæmia, especially eclampsia. Albuminuria is the most important finding of all. When albumin is found, the urine must be examined for the amount of urea in it. If it is much less than the normal 2 per cent. it probably indicates retention of this as well as of other waste products (that should be excreted by the kidneys) in the blood. In suspicious cases the blood ought to be examined for the amount of

urea nitrogen. Normally it is found in the proportion of 0·15 to 0·40 mg. per cubic centimetre of blood. If it is higher than 0·50 mg. per cubic centimetre it is a most ominous sign and treatment must be commenced without delay.

(*e*) *Body weight.*—A sign of some value is increase in the patient's weight out of proportion to the increase due to the progress of pregnancy. This may indicate latent œdema. On the other hand, any cessation in the increase of body weight may indicate death of the fœtus.

The examination of the urine is the most important of all the above signs. This ought to be carried out regularly every four weeks during the whole of pregnancy, and every week during the last few weeks. Primiparæ are more prone to toxæmia in the proportion of 2 : 1. When any of the threatening signs occur, treatment must commence at once along the following lines:—

(i) *Rest in bed*, with hot applications to the back. This helps circulation through, and elimination by, the kidneys.

(ii) *Restricted diet.*—Water only to be taken for the first twenty-four hours and then a diet consisting mostly of carbohydrates, such as fruits, cereals, bread thinly buttered, syrup, chocolate and vegetables. Salt and spices, as well as alcohol, tea and coffee, are to be avoided.

(iii) *The bowels must be made to act freely* by salines such as Glauber's or Epsom salts.

If in spite of these lines of treatment the symptoms do not disappear, or actually increase, it might be necessary to terminate the pregnancy.

Glycosuria.

The urine may also contain sugar. This sugar may be of two kinds, viz., lactose and dextrose. The lactose, or milk-sugar, is present in some 16 per cent. of cases and is of no pathological significance, as it is due to absorption of milk-sugar from the functionating breasts. It must, therefore, be carefully distinguished from dextrose which occurs in diabetes. The distinction is made by means of the fermentation test. Lactose does not ferment with yeast, whilst dextrose does. A full test tube of urine, containing a little fresh baker's yeast, inverted over a cup of water will determine the presence of dextrose by the appearance of gas at the top of the tube. The absence of fermentation after a few hours will show that the sugar found was lactose. The polariscope will also decide, as glucose is dextro-rotatory and lactose is lævo-rotatory, and the osazone test is best of all. If glucose is found, it is not quite safe to diagnose true diabetes, as, owing to the diminished sugar

tolerance of pregnant women as the result of the increase of the pituitary and similar glands, sugar may appear in the urine before the normal threshold of 0·17 per cent. in the blood has been reached. Moreover, the permeability of the kidneys for sugar is increased during pregnancy. Before, therefore, diagnosing true diabetes one must also examine the blood. If the percentage of sugar in it is less than 0·2 per cent., the condition must be considered as renal glycosuria rather than true diabetes.

Such renal cases not only do not require carbohydrate restriction, but are actually exposed to great risk by dietetic treatment, since the blood may be depleted of its sugar to a dangerous degree.

If true diabetes is found, both from urine and blood examinations, as well as from the presence of the classical symptoms, viz., thirst, wasting, polyuria, &c., the matter is of very serious importance. Before the modern treatment of diabetes was introduced 27 per cent. of such women died within twenty-four hours of delivery, and 23 per cent. more within two years. The new treatment with insulin, &c., has made the condition much less serious and many such women are now said to pass through pregnancy and labour in safety. If the condition persists in spite of treatment, induction of premature labour may become necessary, though some believe such treatment to be very rarely required, and to be of very little value.

According to Whitridge Williams, whilst gestation *sometimes* exerts a deleterious influence upon the course of the disease, it *usually* has a beneficial effect upon it, especially towards the end of pregnancy, probably as the result of transmission of insulin from the fœtal pancreas to the mother.

(4) The medical attendant will **measure the pelvis,** and not only detect actual narrowness but will ascertain its size relative to that of the fœtus, and will also detect any pelvic tumours which might obstruct labour.

(5) Lastly, the doctor will detect any abnormal position or presentation of the fœtus and correct it. In doubtful cases X-ray examination should be made (see Plate facing p. 262).

A point worth remembering in connection with the hygiene of pregnancy is the fact that drugs pass from the maternal to the fœtal circulation, and although the fœtus, in virtue of its not breathing independently, is resistant towards morphine, the administration of the drug to the mother immediately before delivery may endanger the respiration of the infant after birth.

State Measures for the Prevention of Maternal and Fœtal Mortality.

In addition to the individual measures to be undertaken by every expectant mother to safeguard herself and her as yet unborn baby, the following provisions must be made for their protection by the State:—

(1) *Registration of abortion and miscarriages*, in order to get reliable information of the fœtal mortality.

(2) *Education in pre-maternity.*—Lessons, by suitably qualified female teachers, on the subjects of the elementary physiology and hygiene of pregnancy should be included in the curriculum of all secondary schools for girls. Also free continuation classes in the same subjects should be provided by every municipal body, and all girls of suitable age should be encouraged to attend them.

(3) *Ante- and Post-graduate Teaching of Ante-natal Physiology and Hygiene.*—Every medical school should have courses of lectures on the subjects of the physiology, pathology and hygiene of ante-natal life in their theoretical and practical aspects.

(4) Medical students, midwives and maternity nurses are to be given a very thorough training in obstetric management and technique, as well as in ante-natal and early post-natal supervision, and a very high standard in these subjects should be expected of all candidates at their respective examinations.

(5) The ante-natal clinics, of which there were 675 (418 municipal and 257 voluntary) in England and Wales in 1925, should have *full-time medical officers* who have had special training in and experience of the various problems connected with the subjects of ante-natal life and practical obstetrics. It is only by making these medical attendants full-time officers that local medical men, who have not either the time or experience to deal with such matters, will be encouraged to send their patients to such clinics. These centres ought also to be attached to the various hospitals, where a sufficient number of *pre-maternity beds* should be available for the purpose of investigating and treating any discoverable abnormality of pregnancy and subsequent labour. During 1923 there were 118 voluntary and municipal maternity homes (excluding hospitals), with 1,442 beds, in which 17,167 patients were treated (Sir George Newman). These, however, provide for confinement only, and do not admit cases for observation. For the 1925 figures see p. 201.

(6) Ante-natal nurses to be sent to expectant mothers who are unable to come to the clinics. These nurses will give general instructions to the mothers, will make periodical examinations of their urine, and arrange for a doctor to make the necessary pelvic and other examinations.

(7) *Pamphlets on ante-natal care* should be sent out to expectant mothers in rural communities. Here registration of pregnancy would afford a very great help in the dissemination of such instructions.

(8) *Public lectures*, illustrated by lantern slides or cinemato-

graphic films, on the hygiene of the expectant mother, &c., should be given, to which mothers, nurses and girls of suitable age should be encouraged to come.

(9) *Special legislation to protect and assist the pregnant woman.*—Under the present National Insurance Acts the maternity benefit consists of a monetary grant of £3 or £1 10s. made to the woman who was delivered of a viable child, according as she is either insured herself or is the legal wife of an insured person. Groaning, as this country is at present, under its heavy load of taxation, it is impossible to expect any Government to increase the maternity benefit in the near future, but under ideal conditions maternity benefit ought to consist of the following :—

(i) Weekly payments to expectant mothers (whether married or not), employed in strenuous gainful occupations, for a specified number of weeks before and after confinement. During this period, which should vary with the nature of the woman's occupation, the expectant mother should not be allowed to work. Also certain industrial occupations should be prohibited to pregnant women throughout their pregnancy.

(ii) Grants in money to provide for the extra expense necessitated by the birth of the baby.

The idea of maternity insurance is not new. Free hospital assistance was given to women in labour in Pfullendorff, Germany, as early as the thirteenth century, and in the fifteenth century, in the same country, financial grants were made to expectant mothers. In France, help was given to nursing mothers in 1317, and in 1796 the French Court decreed that all mothers were entitled to hospital care. In 1891 the Mutualité Maternelle was founded in Paris. In Italy, a society for the benefit of pregnant women was organised in 1752.

At the present day laws for maternity are on the statute books of most European countries as well as some States of America. In Germany no woman may work for four weeks after confinement, and, unless the woman is then certified by a doctor to be in good health, must stay away from work for another two weeks. A similar law prevails in Austria. In Switzerland, which was the first country in the world to pass such an act, in 1877, there is a compulsory rest period for two weeks before and six weeks after confinement. In France the compulsory rest period of eight weeks is equally divided before and after confinement. In this country (England) no woman may be employed within four weeks after childbirth. Similar laws prevail in Italy, Belgium, Portugal, Holland, Norway and Sweden, and in some States of America (Connecticut, compulsory rest of four weeks before and four weeks after confinement; Massachusetts and Vermont, two weeks before and four weeks after; New York, four weeks after confinement). The most extensive—and some would think too extensive—provisions are those made by the Russian Soviet Government. Under their laws pregnant women and nursing mothers must not work in industrial occupations during

the night or overtime. Leave from work, *with full pay*, is granted to such women engaged in manual labour, for a period of four months, equally divided before and after childbirth. If the woman is engaged in intellectual work the compulsory rest period is three months (six weeks before and six weeks after childbirth). In cases of miscarriage, manual workers are allowed three weeks' leave (with full pay), and intellectual workers two weeks' leave. Also, nursing mothers must have half-an-hour's leisure, at intervals of three hours, for nursing their children. Women who are not employed receive a grant of a considerable sum of money for their confinement if their husbands are employed. The wives of those employed in England under the auspices of the Russian Government receive a grant of £15.

(10) Lastly, *prenuptial clinics* should be established where persons of both sexes can get expert advice regarding their fitness to marry, both from the genetic and constitutional, physical and

Fig. 72.—The kind of preparation for marriage formerly given by parents. Some States of America forbid a marriage unless one or both parties can furnish a physician's certificate of freedom from venereal disease.

mental, standpoints, especially with regard to venereal disease and mental disease or predisposition. For it must be remembered that the most important thing in connection with fœtal and infant hygiene is to choose one's parents.

LITERATURE.

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PART III

POST-NATAL HYGIENE

"The hour arrives, the moment wish'd and fear'd;
The child is born, by many a pang endear'd."

ROGERS

SECTION I

NEO-NATAL STAGE

"Nothing so difficult as a beginning."

THE MIDRASH

CHAPTER X

THE PHYSIOLOGY OF EARLY POST-NATAL LIFE

"A lovely Being, scarcely formed or moulded,
A Rose, with all its sweetest leaves yet unfolded."
BYRON.

IN order to understand the principles that govern the hygiene of early life it is necessary to have some idea of the physiological and other characteristics that are peculiar to that period of life. It is convenient to divide infancy into (*a*) *the neo-natal period*, i.e., the first month of life, and (*b*) *later infancy*.

(A) The Peculiarities of the Neo-natal Period.

The neo-natal period is the most critical period in the life of the child. During its intra-uterine existence the child's whole vital economy depends upon a proper interchange of gases and other materials between the mother and itself, and provided no poisonous substances succeed in breaking through the placental barrier, the fœtus lives and thrives, even though it may have certain malformations which are either incompatible with its post-natal existence or minimise the chances of the infant's survival. Thus, a fœtus may have its œsophagus entirely occluded, but because it derives its nourishment not through its alimentary canal but direct from its mother's blood into its own [1] through the placenta, it lives and grows like an ordinary normal fœtus. Directly it is born, however, and it has to depend for its nourishment upon food getting into its stomach through the mouth, it is obvious that any obstruction between the mouth and the stomach will necessarily kill the infant by starvation in the course of a very few days. The same applies to other malformations or monstrosities, such as occlusion of the urethra, absence of brain, &c. Moreover, even in a normal infant, the same

[1] H. G. Wells, in his "War of the Worlds," makes the Martians feed themselves by a modification of the fœtal method of nutrition, viz., by injecting the blood of other animals directly into their own veins.

FIG. 73.—Marked moulding of the skull in occipito-posterior position. The curved line represents the shape of the normal skull. (Von Reuss, after Bumm.)

FIG. 74.—Indentation of skull (parietal bone) resulting from pressure against the promontory of a flat pelvis. (Von Reuss, after Bumm.)

revolutionary change in its mode of nourishment taking place at birth throws a considerable amount of work upon its hitherto unexercised and inactive digestive system. Again, a child born with harelip and cleft palate would, on account of the difficulty of feeding it, have its chances of survival greatly reduced, for

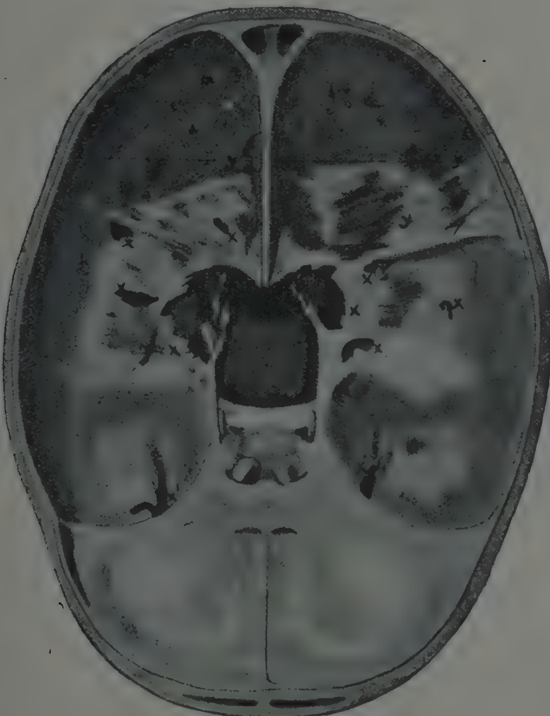

Fig. 75.—Floor of the skull showing injury of the brain membrane at x and y. (Von Reuss, after Bauereisen.)

although it is possible to remedy each defect by a surgical operation, such a procedure is itself accompanied by very considerable risks.

In addition to this, the fœtus in the process of birth goes through a period of stress and strain. The whole body, and

especially the head, becomes compressed, the degree of pressure varying with the relative sizes of the fœtus and the pelvic channels through which it has to pass (see figs. 73, 74, 75). The head may become so compressed as to cause either effusion of blood into the scalp (cephalhæmatoma) or serious injury to the brain. There may also result certain paralyses, either from the intracranial bleeding (Little's disease), injury to the spinal cord in cases of excessive traction in breech cases, or from compression or actual rupture of certain nerves, the so-called obstetrical paralyses, resulting as a rule from faulty obstetrical technique. There may also be bleeding into certain of its internal organs, such as the suprarenals, brain, &c., resulting in intra-natal or early neo-natal death.[1] As we have seen in Chapter IV, p. 131, intracranial injuries are responsible for about half the number of fœtal deaths; such deaths would be more frequent still were it not for the protection afforded to the brain by the architectural arrangement of the intracranial septa of the dura mater, viz., the tentorium cerebelli, the falx cerebri, &c. (Eardley Holland). Moreover, it is only the loosely united bones of the vault of the skull that are subject to moulding; the base of the skull is rigid and does not yield in the process of parturition.

Ylppö[2] investigated experimentally the cause of such hæmorrhages in the new-born. He applied suction cups (connected with a manometer) to the chests of 104 premature and full-term new-born babies, and observed that when sufficient negative pressure was produced in the cup subcutaneous hæmorrhage resulted, and this necessary negative pressure was least in premature and most in well-developed infants, as follows:—

Birth-weight	Hæmorrhage occurred at a negative pressure of
Under 1,000 grm.	150 mm. Hg
1,000-1,500 ,,	250 ,, ,,
1,500-2,000 ,,	310 ,, ,,
2,000-2,500 ,,	400 ,, ,,
2,500-3,000 ,,	450 ,, ,,
Over 3,000 ,,	520 ,, ,,

He therefore explains the intra-natal hæmorrhages into the brain or other internal organs as follows: When the membranes have ruptured, the part of the fœtus which is outside the uterus is subjected to atmospheric pressure only, whilst that inside the uterus is subjected in addition to the intra-uterine pressure, which may be as high as 250 mm. Hg. There is, therefore, a relative negative pressure on the parts outside the uterus equal to the intra-uterine pressure—which, as the above figures show, is sufficient to cause rupture of the blood vessels in under-sized babies.

[1] See H. Spencer, "On Visceral Hæmorrhage in Stillborn Children," *Trans. Obst. Soc.*, 1891, vol. xxxiii.

[2] *Zeitschr. f. Kinderheilk.*, 1924.

"At birth," says Stanley Hall, "the child is cast like a shipwrecked mariner by angry waves on a strange and unknown coast and finds itself in a new and rigorous climate. Instead of the conditions of fluid pressure equal on all sides, it is now in a stereometric world. Its nascent senses are assailed by new stimuli; the alimentary canal now comes into full function; the lungs are first inflated with a gasp. . . . It is not strange, therefore, that the new-born child rarely gains and often loses weight during the first few days. . . . Indeed, with all these profound readjustments the marvel is that the loss is not greater."

During the neo-natal stage the moulded head and body gradually assume their normal shape, the heart and big blood-vessels go through certain evolutionary changes from the types existing in fœtal life to those compatible with independent post-natal existence, and the lungs expand with the first few cries. Should the lungs fail to expand properly (atelectasis) or the heart fail to go through its complete evolutionary post-natal changes, the results to the infant are serious in the extreme.

In addition, the infant is liable to **infection**, either (*a*) *pre-natally*, (i) through absorption of toxins by means of the placental circulation (e.g., cholangitis, resulting in occlusion of bile-ducts), (ii) through the entry of germs into the amniotic cavity in cases of premature rupture of membranes (resulting in stomatitis, parotitis, or broncho-pneumonia); or (*b*) *intra-natally*, through contamination with organisms (gonococci, streptococci, &c.) inhabiting the maternal vagina, resulting in ophthalmia neonatorum; or (*c*) *post-natally*, through the hands of the accoucheur in the process of cutting the cord, resulting in septic infections leading to biliary occlusion, tetanus neonatorum, &c.

No wonder, therefore, that the neo-natal mortality is so extremely high (see p. 143). Indeed, it has been estimated that the chance of a new-born baby to live a week is less than that of a man of 90, and its chance of living a year is less than that of a man of 80. It has further been estimated that the life of a baby at birth is more hazardous than that of a soldier in the trenches, and that its chances of surviving a year are less than that of a professional air pilot. It is by a realisation of the peculiar features of neo-natal life that we may hope to combat the various dangers attending the baby at this early stage of its existence.

Other peculiarities characterising the neo-natal period are certain manifestations which are somewhat analogous to those occurring at puberty. Thus,

in boys there may be swelling of the testicles due to spermatogenetic activity, as shown histologically—sometimes accompanied by serous effusion into the tunica vaginalis—giving rise to a hydrocele, as well as swelling of the prostate; and in girls there may appear a flow of blood from the uterus similar to that occurring at a menstrual period. Also little white pimples of a transitory nature may occur on the skin, due to obstruction of the sebaceous glands, analogous to the acne of puberty. In addition there is sometimes seen in both sexes a congestion of the mammary glands, accompanied by secretion of milk. This commences on the second or third day, and if left alone disappears before the second or third week.

This mammary engorgement is believed to be due to the same cause as the mammary engorgement of the mother, viz., sudden separation from the placental hormones.

Two important phenomena occurring during the neo-natal period are: (1) The initial loss of weight; (2) physiological jaundice. These will be briefly considered in this chapter.

Weight and Body Measurements at Birth.

Birth Weight.—The average weight at birth of a full-term baby is 3,250 grm. (7·15 lb.) in the case of boys, and 3,000 grm. (6·6 lb.) in the case of girls, but many divergences occur, as the following table, given by Hecker, shows :—

TABLE XCVII.

Weight	Frequency
2,500-2,999 grm.	25·8 per cent.
3,000-3,499 ,,	44·4 ,,
3,500-3,999 ,,	24·7 ,,
4,000-4,499 ,,	4·6 ,,
4,500-4,999 ,,	0·48 ,,
5,000-5,499 ,,	0·02 ,,

From this table we see, therefore, that the commonest weight is between 3 and 3·5 kg., with an average of 3·3 kg. Heidemann[1] gives the following table in the case of 360 children :—

TABLE XCVIII.

Weight	Frequency
1,500-1,999 grm.	0·5 per cent.
2,000-2,499 ,,	6·7 ,,
2,500-2,999 ,,	27·8 ,,
3,000-3,499 ,,	40·5 ,,
3,500-3,999 ,,	21·6 ,,
4,000-4,499 ,,	2·9 ,,

[1] *Monatssch. f. geb. u. Gyn.*, 1911, xxxiii, p. 168.

Here the average birth weight works out at 3·17 kg., and the commonest weight is seen to be between 3 and 3·5 kg. Cases of "giant babies" sometimes occur, the biggest recorded having had weights of 11·3 kg. (24·86 lb.),[1] and 12 kg. (26·6 lb.),[2] respectively, but such weights are extremely rare. Indeed, out of 30,000 births, Winckel did not come across one weighing more than 6 kg. (13·2 lb.).

The birth weight varies with race, as the following table shows :—

TABLE XCIX.

Country	Birth weight
America	3,025 grm.
Denmark	3,333 ,,
England	3 410 ,,
Japan	3,013 ,,
Norway	3,466-3,524 ,,
Russia	3,050-3,357 ,,
Spain	3,068 ,,
Sweden	3,527 ,,
Switzerland	3,172 ,,

For the influence of maternal environment upon birth weight see p. 122 *et seq.*

Loss of Weight soon after Birth.—It is an established fact that babies lose weight during the first three or four days after birth. They then begin to regain it, and by the end of the week or so they regain their original weight, after which they gradually increase in weight. Different authorities give different figures for the average nett loss, but there is very little doubt that the loss varies with (1) the amount of the exposure of the infant, as well as (2) the amount of nourishment it receives from its mother. Thus, Professor Louise McIlroy has reduced the initial loss by about 60 per cent. by the simple expedient of substituting inunction with oil for the traditional bath and keeping the baby warm with hot-water bottles (see Chapter XI, p. 312). Also experience has shown that babies of multiparæ, in whom the secretion of milk during the first few days of the puerperium is more abundant, lose less weight than those of primiparæ, whose secretion is somewhat scanty during the first few days (Table C). Premature infants lose absolutely less but relatively to their weight more than full-term babies, but with proper care the loss in them can be reduced to a minimum.

It may be said that in general the loss is equal to about 8 to 10 per cent. of the birth weight, i.e., on the average 250 to 300 grm.

[1] H. Dubois, "Thèse de Paris," 1907, quoted by M. Oyamada, *Zeitr. z. Geb. u. Gyn.*, 17, 1912.

[2] Block, quoted by Gundobin.

(9 to 10 oz.), but a loss of as much as 700 grm. (1½ lb.) has been recorded.

TABLE C.

LOSS OF WEIGHT OF BABIES OF PRIMIPARÆ AND OF MULTIPARÆ (PIES).

Weight at birth in grm.	Primiparæ			Multiparæ		
	Number of children	Loss		Number of children	Loss	
		Net	Percentage		Net	Percentage
2,500	3	240 grm.	11·2 per cent.	4	195 grm.	8·2 per cent
2,510-3,000	20	235 ,,	8·3 ,,	8	180 ,,	6·2 ,,
3,010-3,500	50	295 ,,	9·0 ,,	15	265 ,,	8·1 ,,
3,510-4,000	31	360 ,,	9·7 ,,	10	325 ,,	8·7 ,,
4,010-4,500	4	245 ,,	8·4 ,,	5	366 ,,	8·3 ,,
Average of	108	300 grm.	9 per cent.	42	270 grm	8 per cent.

Causes of Loss of Weight.—The loss is due to two main causes: (1) *Mechanical*, viz., loss of urine, meconium and vernix caseosa, &c.; (2) *Physiological*, viz., the actual loss of body tissue—*principally loss of water* from the lungs and skin. This water loss has been measured and found to be 28 grm. per kg. body weight for the first twelve hours and 40 to 54 grm. per kg. during the subsequent forty-eight hours. The combined loss sustained in these ways is more than is compensated for by the amount of nourishment received from the scantily secreting breasts of the mother (see "Child Physiology," pp. 278 and 423).

Prevention of Loss of Weight.—Theoretically it should be possible to prevent this initial weight loss, (1) by minimising the loss of heat and tissue waste of the baby, by anointing it and keeping it very warm; (2) by giving the baby a little extra nourishment in the form of sugar water. Statistics, however, have shown that the loss is not detrimental, since babies whose loss has been thus minimised or avoided have, after a few weeks, shown no advantage over those in whom loss of weight has been allowed to take place in the usual way. Indeed, even babies who have lost as much as 500 grm. (17½ oz.) have subsequently developed in a perfectly normal manner.

Birth Length.—The average length of a baby at birth is about 50 cm. (20 in.), but it varies from 47·5 cm. to 54 cm. in boys and from 43 cm. to 53 cm. in girls. Unusually large babies, however, are sometimes met with, and a birth length of 75 cm. (30 in.) has been recorded. The length does not show any

diminution during the first few days corresponding to the loss in weight, although babies that show marked moulding of the head may become a little shorter during the time the head returns to its normal shape.

The coefficient of correlation between the birth weight and the birth length in male infants is 0·644 ± 0·012 (Pearson).

As regards the *other body measurements*, the following are the peculiarities of the new-born baby: (1) The sitting height is 66·6 per cent. of the total length; as the child grows the proportion becomes less. (2) The circumference of the head is slightly greater than that of the chest, in the proportion of about 14 : 13·5 in.; it becomes equal to it at one year (18 in. each), after which the chest circumference exceeds that of the head.

Icterus Neonatorum.—A large number of infants show a varying degree of jaundice of the skin, starting a couple of days after birth and disappearing during the second week. This jaundice is characterised by the fact that the stools retain their normal colour (because they are not deprived of bile pigment), and the urine is free from bile, as tested by its colour, the non-staining of the napkins, and by the usual chemical tests for bile pigments. It is these characters of the stools and urine which distinguish this *simple* or *physiological jaundice* from the other serious or pathological types, such as that due to congenital obliteration of the bile ducts, &c. According to Ylppö, this physiological jaundice, which cannot be prevented, and requires no treatment, because it always disappears in a few days, occurs in about 80 to 85 per cent. of all babies and in practically 100 per cent. of premature infants.

The **cause** of this condition is not at all well known. Many theories have been suggested but not one of them is entirely satisfactory. Till recently it was believed that all bile was formed (out of the red blood-cells) *in* the liver, and that in the case of the new-born baby, the liver being imperfectly developed, they are unable to drive all the bile pigment into the bile canaliculi, and thence into the bile duct and into the duodenum, and therefore some of the bile is passed on to the blood, giving rise to the jaundice. For this reason premature infants in whom the liver is still less well-developed are more liable to jaundice. Recent work, however, has shown that bile pigments can be formed from broken-down red blood-corpuscles by special cells—called the reticulo-endothelial system—*outside* the liver, e.g., in the spleen, &c., and that the main function of the parenchymatous liver cells is to transform the bile pigments thus formed from one form into another, which can be distinguished by the different kinds of reaction that the two forms give with Ehrlich's diazo reagent. Bile pigment, before its passage through the liver cells, gives the so-called *indirect* reaction, whilst after the action of the liver cells upon them they give the *direct* reaction. Now it has been found that

the blood-serum in cases of physiological icterus neonatorum gives the indirect reaction, showing that the bile pigments have not passed through the liver cells and have been derived from the hæmoglobin formed from broken-down red cells. These direct and indirect reactions are called, after their discoverer, the van den Bergh tests.

The point in favour of this theory is that the red blood-cells of the new-born are more fragile than those of the adult, and of the premature infant more fragile than those of the full-term one. The point against it is that there is no real evidence that red cells are actually being broken down in early post-natal life. Also, the author has published a case where the indirect reaction was given by pigment *after* its passing through the liver cells.[1]

From the **pathological** point of view, the neo-natal period is characterised by the following: Hydrocele neonatorum; mammary engorgement; cephalhæmatoma, obstetrical paralysis (of the brachial plexus or of the facial nerve), umbilical infections, ophthalmia neonatorum and congenital malformations. These are considered in books dealing with obstetrics or the diseases of children. Ophthalmia neonatorum is dealt with in the next chapter (p. 315).

(B) The General Peculiarities of Infant and Child Life.

The infant differs from the adult in many respects, as follows:—

(1) *Anatomical.*—A cursory comparison of the skeletons of the adult and the new-born infant is sufficient to convince the most casual observer of the enormous differences that exist between them. The head, spine, thorax, pelvis and limbs of the skeleton show marked differences, both macroscopically and microscopically, and chemically as well as physically, so that an infant must not be considered as merely "a man in small letters." He is an *immature* and not merely a miniature adult. Imagine for a moment a toothless adult with a head so large that it forms a quarter instead of the normal eighth of the whole length of the body; whose legs are another three-eighths instead of the normal half the body length, and whose liver is about twice the normal size. Imagine further that the vault of his skull, instead of being a perfectly rigid box, consists of bones movable one upon the other, with spaces between the adjacent bones (fontanelles) through which the pulsations of the brain can be felt. Such an individual would indeed be a monstrosity! Yet such is the state of things in the normal condition of an infant at birth (fig. 76).

(2) *Physiological.*—The numerous physiological peculiarities may be summarised as follows:—

[1] W. M. Feldman, "A Case of Congenital Occlusion of the Common Hepatic Duct in a Twin Baby with an Indirect van den Bergh Reaction," *Lancet*, 1924, ii, 113.

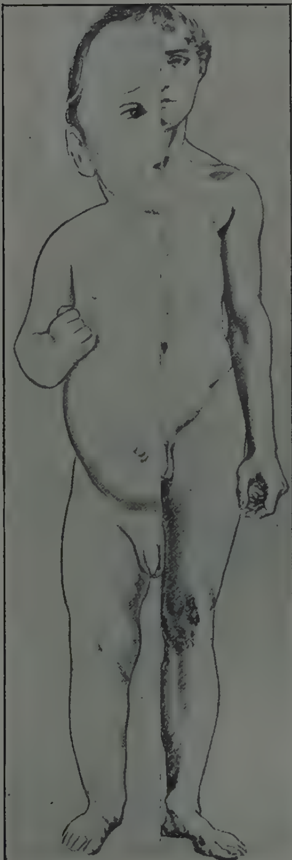

FIG. 76.—The right side of a child—magnified to the size of an adult—placed in contact with the left side of an adult to show the difference in proportion of the various parts. (The illustration was constructed from the pictures of a child and an adult, forming the frontispiece to Forsyth's "Children in Health and Disease." London: John Murray, 1909.)

(i) The child is a *growing animal*, and therefore requires extra food and calories (called *the growth quotient*) over and above those required by the adult to replace wear and tear of tissue. This growth quotient has been estimated to be about 13 per cent. of the ordinary requirements (see p. 356).

(ii) *The surface area of the body is relatively larger, per unit body weight*, in the child than in the fully-grown adult. This is a geometrical law applying to all bodies of different sizes, which says that small bodies have larger surfaces in relation to their bulk than have large bodies of the same shape. The law will be understood from an examination of the diagram (fig. 77), in

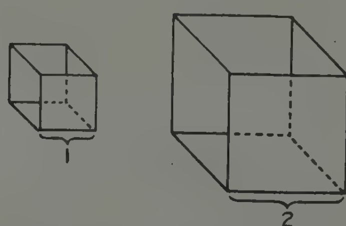

FIG. 77.—Diagram to illustrate relation between volume and surface in bodies of different size.

which it is seen that the total areas of the six surfaces of the smaller cube, whose side is *one* unit, is *six* square units, and its volume or weight is *one* cubic unit, so that the area of its surface per unit weight = 6 square units. But in the case of the larger cube, whose side is two units, the total area of the six surfaces is $6 \times 4 = 24$ square units, and its volume is 8 cubic units, so that the area of its surface per unit weight $= \frac{24}{8} = 3$ square units. In other words, the surface area per unit weight (i.e., *the specific surface*) of the smaller cube is twice as great as that of the larger cube. There are several formulæ by means of which the exact surface of the body can be calculated in any individual; one is $S = K W^{\frac{2}{3}}$ (where S = surface, W = weight, and K = a constant). This is Meeh's formula. Another formula is the Du Bois height-and-weight formula. This, as well as the Feldman and Umansk chart for using it rapidly, is given in the diagram (fig. 78).

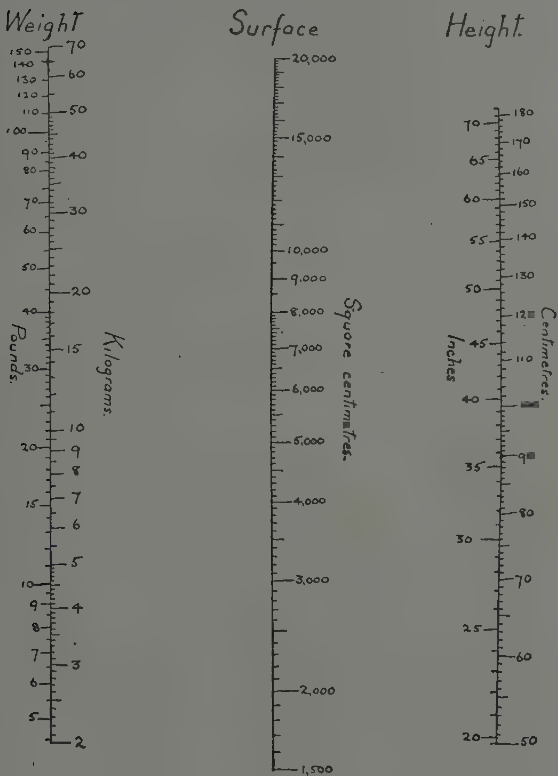

FIG. 78.—The Nomogram for the Du Bois formula : $S = 71{\cdot}84\ W^{0.425}\ H^{0.725}$, where S = surface in sq. cm., W = weight in kg. and H = height in cm. A straight line joining given values of W and H cuts the middle scale at the correct value of S. Thus a line joining the points 24 on the weight scale, with the point 110 on the height scale, will cut the surface scale at a point corresponding to 8375, which means that the surface area of a person 24 kg. in weight and 110 cm. in height is 8375 sq. cm. (Feldman and Umanski.) (*Reproduced from the "Lancet."*)

The following table (Table CI) illustrates some of the results found :—

TABLE CI.

Age	Body weight	Body length	Body surface: Dubois formula: Total	Body surface: Dubois formula: Per kg.	Body surface: Formula $S = K W^{\frac{2}{3}}$ *: Total	Body surface: Formula $S = K W^{\frac{2}{3}}$ *: Per kg.
	kg.	cm.	sq. cm.	sq. cm.	sq. cm.	sq. cm.
At birth	3·2	52·5	2,100	656·3	2,171	678·5
1 month	3·8	55	2,300	605·3	2,435	640·8
3 months	5·0	60	2,800	560·0	2,924	584·8
6 ,, ...	6·5	63	3,200	492·5	3,482	551·8
1 year	9·6	72	4,200	437·5	4,788	498·7
2 years	13·0	90	5,600	430·8	5,861	450·8
10 ,, ...	28·0	130	10,000	357·1	10,603	378·7
Adult	70·0	172	18,000	257·1	19,527	278·9

* *Note.*—The value of K is, according to Benedict and Talbot, different for the two sexes and for various weight intervals as follows :—

TABLE CII.

Boys: Body weight	Boys: K	Girls: Body weight	Girls: K
Up to 6 kg.	10·0	Up to 6 kg.	10·0
6-15 kg	10·6	6-10 kg.	10·6
15-25 ,, ...	11·2	10-20 ,, ...	10·8
25-40 ,, ...	11·5	20-40 ,, ...	11·1

The following table (Table CIII), which is easy to remember, is sufficiently accurate for practical purposes :—

TABLE CIII.

Weight	Surface	Body surface per kg. body weight
3 kg.	2,000 sq. cm.	650 sq. cm.
6 ,,	3,000 ,,	500 ,,
9 ,,	4,000 ,,	450 ,,
12 ,,	5,000 ,,	400 ,,
15 ,,	6,000 ,,	400 ,,

In other words, for every 3 kg. increase in weight of the child there is an increase of 1,000 sq. cm. in its surface area.

From Table CI it is seen that the infant at birth has a

surface area, per unit body weight, about two and a half times as large (i.e., $\frac{6.563}{2.571} = 2.6$) as has an adult. The significance of this is disputed. According to Rubner and his school, the loss of heat by the body is proportional to its surface area, and hence a young infant loses about twice as much heat for each unit of its weight as does an adult. This means that not only cannot young infants stand exposure to cold as well as adults, but that they require food of particularly high calorific value, such as *fat* (whose combustion yields 9 calories per grm., as against 4 calories per grm. yielded by the combustion of either protein or carbohydrate in the body). Indeed, if we compare the average diet per day of an ordinary adult with that of an infant, we get the following results :—

TABLE CIV.

	Average adult weighing 70 kg.	Infant at 6 months weighing about 7 kg.
Protein	118 grm. = 1·9 grm per kg.	14 grm. = 2 grm. per kg.
Fat	56 „ = 0·8 „ „	30 „ = 4 „ „
Carbohydrate ..	500 „ = 7 „ „	59 „ = 8·5 „ „
Calories	3,054 „ = 43·5 „ „	578 „ = 82·5 „ „

So that whilst in respect of protein and carbohydrate the infant receives approximately the same quantities as the adult per unit of body weight, it receives relatively five times as much fat and twice as many calories as does the adult.

According to the recent and very exact researches of Benedict and Talbot, however, this "body-surface law" does not hold good. They attribute the relatively greater loss of heat by the infant to the greater proportion of active protoplasm in its cells.

The matter is not quite settled, but assuming Rubner's theory to be correct, we can see how a knowledge of the surface area of any given infant can be utilized for the purpose of ascertaining the amount of food required by it.

Supposing it to be known that the daily amount of heat lost by the body is 1,500 calories per square metre of surface, and we want to know how much cow's milk, whose calorific value is about 730 calories per litre, an average infant weighing 5 kg. (11 lb.) and whose length is 60 cm. (24 in.) requires in twenty-four hours, we proceed as follows :—

The surface area of such an infant is 0·28 square metres (see fig. 78). Therefore the daily number of calories lost by it is equal to $0·28 \times 1500 =$ 420 calories.

But the growth quotient, i.e., the extra amount of calories required

for growth is, as we have seen, 13 per cent. $= \frac{13 \times 420}{100} =$ approximately 60 calories. Therefore the total number of calories the infant requires $= 420 + 60 = 480$ calories.

But calorific value of milk = 730 calories per litre or 20 per oz. Therefore amount of milk required by the baby $= \frac{480}{730} = 0{\cdot}66$ litre or $\frac{480}{20} = 24$ oz.

The heat produced or lost by any particular baby is by no means constant. It varies a great deal with the amount of muscular activity of the infant—the greater the activity or crying, the greater the amount of heat lost—as well as with the amount of clothing the baby wears. On the average, however, it is not far from the truth to say that *up to about six months, the heat loss of an infant is about* 50 *calories per pound weight, and gradually diminishes so that at one year the heat loss is no more than about* 45 *calories per pound.*

(iii) *The Digestive Apparatus is Immature and its Functions are Imperfect.*—The infant has no teeth, and must therefore be given its food in liquid form, and the various digestive glands are not yet fully developed. This is especially so with regard to the amylolytic or starch-splitting ferments. It is, however, incorrect to say that young infants cannot digest starch at all, since small amounts of the ferment have been demonstrated in the saliva and pancreatic juice not only of new-born infants but even in fœtuses, and hence small quantities of starch, such as barley water, may cautiously be given even to very young infants.

The various intestinal secretions such as secretin, enterokinase, invertin, erepsin, &c., have been found at birth, and bile begins to flow two hours after birth. An important point in connection with the young infant's intestinal tract is that the mucous membrane cannot properly deal with foreign proteins such as cow's caseinogen or egg white. This is especially the case with new-born babies and with infants suffering from some gastro-intestinal disturbance. When such infants, therefore, are given cow's milk or white of an egg, these foreign proteins fail to be entirely broken up in the intestine and may be absorbed unaltered into the blood-stream, where they act as sensitisers, rendering the infant susceptible to the toxic action of the proteins absorbed after an interval of more than twelve days. This phenomenon of increased sensitisation is called anaphylaxis, and it is possible that many conditions in infants, such as skin irritation, asthma, &c., may be mildly anaphylactic in nature. Hence the common practice of giving albumin water to young babies suffering from gastro-enteritis seems to be unscientific,

because, unless the egg white is given continually after that, the infants may become sensitised to egg white and be unable to take eggs later on without developing rashes. Sugar water is a better, more nutritive and more scientific substitute.

The Nem Method of Feeding.

Another interesting point, of more than purely academic value, is the fact that the total length of the small intestine of any person at any age, whether infant, child or adult, bears a constant relation to that person's sitting height. Indeed, it has been shown to be equal to about ten times the person's sitting height. It has also been shown that the average circumference of any person's small intestine is one-tenth of the sitting height. But the total area of the intestine is, like the total area of any cylinder, equal to length × circumference. Therefore, any person's intestinal area = 10 Si $\times \frac{1}{10}$ Si = $(\text{Si})^2$; (Si = sitting height). Hence, by measuring any infant's or child's sitting height, we can, by squaring it, find the total absorptive area of its intestine. Now, von Pirquet finds that in the same way as there is an external surface area law (see Rubner's law above), so also is there a law of internal surface area. In fact, he finds that a growing infant under six months old requires a number of calories equivalent to ½ c.c. of milk for every square centimetre of its intestine. Hence, by the simple process of measuring a baby's sitting height we can find how much food it requires per diem. Thus, if the sitting height of a baby is 35 cm., then its food requirement is $\frac{1}{2}(35)^2 = 613$ grm. of milk, or about 22 oz.

This method of feeding, called the Nem method (Nahrungs Einheit Menge or nourishment unit amount), is based upon the further supposition that provided an adequate amount of protein is given, the balance between carbohydrate and fat is of no importance so long as the necessary number of calories are given during the day. These calories may, according to Pirquet (who has tried this method in Vienna during the war, when fat was scarce and expensive), be made up entirely by means of carbohydrate. (For Comments see Chapter XIII, p. 365.)

Free HCl, as well as pepsin, rennin and lipase, are found in the stomach at birth. The concentration of the acid, however ($C_H = 0{\cdot}005$), is probably not enough for the pepsin. The pancreas, in addition to its amylopsin, also contains trypsin and steapsin. The presence of all the salivary, gastric, pancreatic and intestinal secretions at birth shows that the infant is equipped, in however slight a degree, with all the secretions necessary to digest starch, protein and fat.

The stomach has a capacity of 1·2 oz. at birth, but this anatomical capacity is no guide to its physiological capacity or to the amount of milk the child can take at a feed, since food begins to leave the stomach (into the duodenum) as soon as it enters it. Hunger contractions (see p. 388) begin two hours after

a meal and reach their maximum in three hours. The gastric capacity at one month is 1·5 oz.

(iv) *The circulation of the infant differs qualitatively as well as quantitatively from that of the adult.*

(*a*) **Qualitative Differences.**—The various structures belonging to the fœtal circulation, viz., the foramen ovale, the ductus arteriosus, and the ductus venosus do not become obliterated till a variable period after birth (although these apertures gradually diminish in size towards the end of pregnancy), and their functional persistence constitutes what is termed "congenital heart disease." There are many other structural differences which it is not necessary to enumerate here, but there is one point in connection with the musculature of the heart which may be referred to in this place. In the adult the left ventricle is a good deal thicker than the right, but in the young infant the two ventricles are of equal thickness. This is a matter of considerable importance to the infant, because in cases of pneumonia or pulmonary congestion, to which infants are somewhat specially liable, the outlook depends upon the state of the right ventricle, and a thick right ventricle is not so likely to fail. This fact, therefore, renders the prognosis of pulmonary troubles considerably less serious than it might otherwise be. This feature also explains the peculiar character of the young infant's electrocardiogram (see "Child Physiology," p. 445).

TABLE CV.—SHOWING THE QUANTITATIVE CIRCULATORY DIFFERENCES BETWEEN INFANT AND ADULT.

	Adult	New-born infant
Pulse-rate per minute	72	120
Blood-pressure in millimetres of mercury	120	60
Circulation time in seconds.. ..	22	12
Output of heart in cubic centimetres	66	7·5
Amount of blood in cubic centimetres circulating per minute per kilogramme body weight	62	310
Areas of cross-section of aorta per square centimetre of body surface	0·026 sq. mm.	0·022 sq. mm.
Work of heart—		
(1) Per beat	160 grm. metres	10 grm. metres
(2) Per day	17,260 kg. metres	1,050 kg. metres
(3) Per gramme heart muscle..	65 ,, ,,	80 ,, ,,
(4) Per kilogramme body weight per day	246 ,, ,,	608 ,, ,,
(5) Per square metre body surface per day	10,000 ,, ,,	9,000 ,, ,,

(*b*) **Quantitative Differences.**—These depend to some extent upon the qualitative ones. Here it would be impossible to show the correlation between the two, and the above table (Table CV), enumerating some of the more quantitative differences, will suffice.

From this table we learn that the infant's circulation is much more rapid and its blood-pressure lower than in the adult, but that *the amount of work done by each unit weight of heart muscle per unit of time, as well as the amount of work done by the whole heart per unit surface of body per unit of time, is practically the same in both infant and adult.*

(v) *The Respiration is Different in Character and Rate.*—The infant's respiration is always abdominal in type, whilst that of the adult is said to be different in the two sexes (abdominal in men and thoracic in women). It is also irregular, and Cheyne-Stokes breathing is not uncommon. The following table (Table CVI) enumerates some of the more important quantitative differences:—

TABLE CVI.—SHOWING THE QUANTITATIVE RESPIRATORY DIFFERENCES BETWEEN INFANT AND ADULT.

	Adult	New-born infant
Respirations per minute	16	44
Tidal air in cubic centimetres	450	48
Vital capacity in cubic centimetres ..	3,500	Undetermined
Amount of air (in cubic centimetres) breathed per minute—		
(1) Per kilogramme body weight ..	120	400
(2) Per square metre of surface of body	5,500	5,700

This table teaches us that the infant's respiration is more rapid, but that *the amount of air breathed per unit of time per unit surface of body is practically the same in both infant and adult.*

As regards vital capacity, it has been shown by Dreyer that it can *in any individual* be correlated with the sitting height, and as the latter can be correlated with the body surface, therefore we have again the result that *the vital capacity per unit surface of body is the same in both infant and adult.*

Another point is the narrowness of the larynx, which renders the child particularly liable to laryngeal obstruction should it have any catarrhal congestion of the throat which narrows the glottis still more. There is a condition called *congenital*

laryngeal stridor, which is probably due to extreme congenital narrowness of the larynx. The condition disappears as the opening of the glottis becomes wider with growth.

(vi) *The character and the relative amounts of blood are different, as shown in Table CVII*:—

TABLE CVII.—SHOWING QUANTITATIVE DIFFERENCES IN THE BLOOD OF THE INFANT AND ADULT.

	Adult	New-born infant
Total amount in body:—		
(1) Per kg. body weight	90 c.c.	150 c.c.
(2) Per square metre of body surface..	Probably the same in both cases (Dreyer and Ray)	
Number of red corpuscles per cubic mm. of blood	5,000,000	6,000,000
Nucleated red cells	None	A few
Total whites	8,000	20,000
Polymorphonuclears	65 per cent.	75 per cent.
Lymphocytes	30 ,,	16 ,,
Eosinophiles	3 ,,	2 ,,
Hæmoglobin	100 (standard)	130
Colour index (i.e., amount of hæmoglobin in each red cell)	1 (standard)	1·1
Blood sugar	0·1 - 0·12 per cent.	0·088 per cent.

These numbers vary greatly at different ages, but the interesting point is that *the total amount of blood in the body, as well as the total amount of hæmoglobin (i.e., the oxygenating part of the blood), is per unit area of the body the same in infant and in adult.*

Although the percentage of polymorphonuclears, which are the phagocytes that destroy bacteria in the blood, is high at birth, it falls immediately afterwards, and indeed between nine and twelve months it is no more than about 36 per cent. (as compared with the 65 per cent. in the adult). It is possibly in virtue of this fact that bacteræmia is more frequent in early infancy than in adult life. Another possible factor is the greater acidity of the serum of an infant's blood. Infection would for this reason be a very much commoner occurrence in early infancy than it is were it not for the fact that the deficiency in polymorphonuclear cells is counterbalanced at that age by the presence of immune bodies retained from the placental circulation, as well as, in the case of breast-fed babies, those swallowed with the mother's milk. It is during the ages between 2 and 6 years, when the polymorphonuclear cells are still below the adult level and the immune bodies acquired from the mother have become exhausted, that the common infectious diseases become most prevalent.

(vii) *The Characters of the Excretions, Urine, Fæces, &c., are Different.*—The infant excretes more water and CO_2 per kilogramme body weight than the adult. This apparently excessive

disintegration of tissue is again to be explained by the law of surface area. This explains why any disturbance of digestion or assimilation has such a profound effect upon growth and nutrition in an infant.

(viii) *The chemical composition of the body, its metabolism and its nervous system are different in the infant from what they are in the adult* (see Chapter XIII, "Child Physiology," Chapter XXI).

In addition, there are also the following other peculiarities, viz. :—

(3) *Pathological.*—The diseases occurring in early life are in many respects different either in character or in distribution from those occurring in the adult. Thus, the separation of the cord renders it peculiarly susceptible to sepsis from that source. Obstetrical paralyses necessarily occur only in early infancy. The same applies also, of course, to congenital malformations, incompatible with life. Rickets only occurs during infancy and early childhood. Tuberculosis, whilst it attacks the apex of the lung in the adult, affects the bones, joints, glands, peritoneum and brain in the child. Digestive disturbances and diseases of nutrition occur chiefly during the first year. Rheumatism usually affects the joints in the adult, but it frequently attacks the brain (St. Vitus' dance) in the young child. Also retropharyngeal abscess, intussusception, convulsions, tetany, and several other diseases occur mostly in infancy.

(4) *Clinical.*—Symptoms have not the same significance in infancy that they have in adult life. Pyrexia, i.e., a rise of temperature, for instance, is not necessarily important in the infant, because the slightest disturbance will upset its imperfectly-developed heat-regulating mechanism. Vomiting and convulsions are as a rule comparatively unimportant in babies, because both the digestive and nervous systems are not yet strong enough to resist ordinary irritation which could easily be resisted by an older individual. The peculiar kind of breathing called *Cheyne-Stokes respiration*, which is almost invariably fatal in the adult, is, on account of the sluggish activity of its respiratory centre, of very little importance in the young infant. Then again, in virtue of the differences in the numbers and characters of the various corpuscles in the bloods of infants and adults, it is clear that blood-counts, which are so important in many diseases, must be interpreted differently in the two classes of patients. Indeed, even the appearance of nucleated red cells, which is of such grave importance in the adult, is of no such importance in very early neo-natal life. A Babinski phenomenon (i.e., an upward, instead of a downward, movement of the big toe resulting from a tickling of the sole of the foot) indicates definite organic disease of certain portions of the nervous system in the adult, but may have absolutely no pathological significance whatever in infants in whom the phenomenon is very frequently present normally.

Further, owing to the greater permeability of the lymphatic vessels in the infant and child, diseases which in adults usually remain localised frequently spread to the neighbouring lymphatic glands in the child. Thus, a mild nasal or pharyngeal catarrh often causes enlargement of the cervical glands. Similarly, the frequency of hilar tuberculosis (i.e., tuberculosis of

the glands at the root of the lungs) in children, with only slight traces of disease at the site of entry of the tubercle bacillus at some part of the respiratory tract, is another example of the same phenomenon.

Again, in virtue of its being in a state of active growth and development, certain diseases of the endocrine glands have a more profound effect on the infant and child than on the adult. Thus, insufficiency of thyroid secretion in the adult results in myxœdema manifesting itself by a certain amount of apathy, dryness and infiltration of the skin, &c., whilst the same deficiency in the young child gives rise to cretinism manifesting itself by a cessation of physical and mental growth. Similarly, pituitary disease gives rise to acromegaly in the adult manifesting itself by localised hypertrophy, chiefly of the soft parts, because the various bony epiphyses have already united, but in the young child, in whom the skeleton goes on growing, such a lesion causes exaggerated skeletal growth resulting in gigantism.

On the other hand, there are certain diseases which are extremely rare or unknown in early life. These are the degenerative diseases, e.g., cancer, arterio-sclerosis, atheroma, chronic interstitial nephritis, &c.

From what has been stated in this chapter it will be appreciated that although the physiology of the infant differs so markedly from that of the adult, the differences are more apparent than real. *It is almost certain that when reduced to a common denominator, i.e., when expressed in terms of the same unit, the various activities of the infant and adult or indeed of any normal individual at any age, are quantitatively the same.* It is not possible to dogmatise what that unit is; unit of body surface, as we have seen, answers the purpose fairly well, but it is possible that the unit might be found to be the unit amount of protoplasm which is in itself a function of the surface.

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CHAPTER XI

NEO-NATAL HYGIENE

"From the instant of our birth we experience the benignity of Heaven and the malignity of corrupt nature."—JOHN TRUSLER.

IN the chapter on "Fœtal Physiology" (Chapter VIII) we saw that the fœtus lived inside the womb floating about entirely submerged in a pool of water at constant body temperature. The liquor amnii constitutes not only a water bath of the same temperature as the blood, but it also acts as a water cushion, protecting the fœtus from all external injuries, microbes and shocks. Inside the uterus also, the fœtus received its life-giving oxygen direct from the mother's blood into its own, and from the same source it received straight into its own blood all the various nutritive materials already suitably split up and prepared by the agency of the mother's digestive apparatus. Further than that none of the waste products manufactured by the fœtus exercised any appreciable strain upon the fœtal excretory organs, such as the kidneys, lungs, bowels, and skin, but were transferred straight from its own blood into that of the mother for the excretory organs of the latter to get rid of them.

During the process of birth the fœtus goes through a series of mouldings to which it is utterly unaccustomed and which compress some of its vital physiological mechanisms. Indeed, were it not for the wonderful architecture of the membranes which line the inside of the skull and form a protective covering to the brain, few fœtuses would survive the strain of being born. Immediately the baby emerges out of its own hydrosphere into our atmosphere it is suddenly assailed by the various stimuli of which it previously had no experience, viz., change of temperature, to which it has to accommodate itself by the agency of its own previously inactive thermotactic apparatus, contact with strange objects, such as the accoucheur's hands, various atmospheric organisms, &c.

With the baby's first cry its lungs are suddenly inflated, and this establishment of pulmonary breathing, together with the

tying of the umbilical cord, causes a profound change in its circulation. The various fœtal characters of the heart suddenly cease to function and the circulation assumes the features characteristic of the adult. The baby now assumes an independent existence. The oxygen it requires must come from the air through its own lungs into its blood; the various nutritive materials must now be passed through its own alimentary tract for digestion and transformation into substances capable of absorption into its blood; and finally its own kidneys, skin, lungs and bowels now have to do the work of getting rid of waste products which before birth were undertaken for it by the excretory organs of its mother.

Owing to these tremendous changes of environment the neonatal mortality is so dreadfully high (see Chapter V, p. 143, *et seq.*). To reduce this suitable measures must be taken, not only ante- and intra-natally to ensure its safe and easy birth but immediately after birth to get the new-born infant gradually and gently accustomed to its new surroundings.

The Baby's Immediate Care.

(1) **Protection against Cold.**—The room in which labour takes place must be suitably warmed and free from draughts. The accoucheur's hands must be warm, and the baby immediately it is born must be well wrapped in a warm blanket. As soon as the cord is tied and cut the infant must be placed in a warm cot containing a hot rubber water bottle over which a soft flannel is placed and in which the baby is wrapped. Great care must be taken that the baby is sufficiently far and protected from the bottle not to be burned. We have seen that owing to its comparatively large body surface, as well as to the imperfection of its thermotactic mechanism, a baby cannot stand frequent exposures to temperatures below body temperature, and hence a baby should be handled with the greatest care. It must not, unless it is suitably covered, be taken from one room to another.

There are some who advocate the hardening process of infants from the moment of birth. Dr. Leonard Hill, who is an ardent believer in the value of cold fresh air, would begin exposure of infants to cool air immediately they are born. In support of his contention he brings forward the following arguments[1]:—

(i) Kittens may be restored after prolonged immersion in cold water.

[1] See *Maternity and Child Welfare*, iii, 1919, pp. 151 and 154.

(ii) The blacks of Australia allow their infants to remain naked from birth, and yet they are a virile race.

(iii) The mothers in Tierra del Fuego (according to Darwin) stand naked in the cold sleet with the naked babes in their arms, and that such exposure does not kill the infants is evidenced by the fact that the Tierra del Fuegians practise infanticide to keep the population down to the level of the food supply.

(iv) He quotes an anonymous report from the *Cape Times* (which may or may not be true) of a new-born infant that was found alive on the veldt after it had been exposed naked for sixty-four hours, during which time it had endured a frosty night, a day of hot sunshine and a day of rain.

"Europeans," says Hill, "unconsciously practise infanticide through the condition of tenements, and in place of quickly strangling their children expose them to massive infection (brought on by lack of adequate cooling and evaporative power of the atmosphere), and reduce their nutrition and resistance to disease."

This is a very strong indictment against the teaching to keep new-born babies particularly warm. I believe, however, that Dr. Hill's arguments, though forceful, are unconvincing. It is true that kittens and animals—including some infants—will stand exposure to cold with impunity, but we have no figures to state what proportion of such exposed animals succumb. The Spartans, as we have seen, practised similar hardening methods, but they only built up a virile race through the elimination of all but the strongest.

The fact that the Tierra del Fuegians practise infanticide to keep the population down only proves that the number of infants that naturally survive in each family is more than they can afford to rear, but it does not tell how many infants survive such exposure. Moreover, what applies to one race that has been hardened by the survival of the fittest, does not necessarily apply to other races living under conditions of civilisation. It must be admitted, however, that judicious and *gradual* hardening, such as accustoming the baby to a cool bath, &c., is without a doubt conducive to good health, but it would be dangerous to try this on babies that are either too young or too feeble. (See p. 313.)

(2) **The Cleaning of the Baby.**—Custom has from the times of antiquity made the bathing of the infant immediately after a birth a law of neo-natal hygiene. This practice, however, is not altogether without its risks. The transfer of the infant to

and from the bath is likely to be attended by a loss of heat, which, apart from its risk of causing chill, involves an extra amount of metabolic activity on the part of the infant resulting in loss of weight. Moreover, the bath water may convey infection to the eyes, navel, and various natural apertures, and also the soap may cause eczema and various skin eruptions. For this reason it is preferable, especially in somewhat weakly babies, to do away with the bath until the baby is somewhat older, at any rate until the umbilicus is healed. Professor Louise McIlroy has treated a number of new-born babies by anointing them with

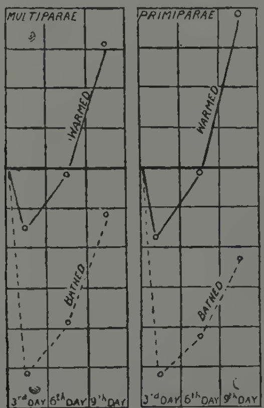

Fig. 79.—Charts showing the average loss of weight in 40 oiled and warmed babies and 40 bathed babies of multiparæ, and 40 oiled and warmed babies and 40 bathed babies of primiparæ.

olive oil, instead of by bathing them, with very satisfactory results. The baby is oiled every day for the first ten days or a fortnight. The oil not only cleans the baby but also, on account of its bad thermal conductivity, keeps the baby warm. The beneficial effect of this method of cleaning a baby as compared with bathing is demonstrated by the above charts, which show that the initial loss of weight is less and the return to birth weight takes place sooner in the oiled than in the bathed babies.

The Bath.—After the cord has separated, the baby, when born maturely, may be bathed. The room temperature should be 70° F. and the bath water should have a temperature of 100° F.

The baby should be rubbed as little as possible in order to avoid rubbing off its delicate skin and causing dermatitis.

The soap should be of the best variety, e.g., Castile soap, Vinolia baby soap, Pears' soap, Lanoline soap, &c., as the cheaper soaps contain excess of alkali which irritates the delicate skin. It is probable that the intense redness as well as the pustular eruption from which many babies suffer during the early neo-natal period are due to friction with irritating soaps.

After the bath, which should last between three and five minutes, the baby is thoroughly, but very carefully and gently, dried

FIG. 80.—Folding bath.

by sponging with a soft towel and not by rubbing. Special attention is to be paid to the drying of those portions of the body like the armpits, groins and buttocks, where two folds of skin are in contact. Dusting powders, especially those containing boracic acid, irritate the skin and should be avoided. If dusting powders are used they should be non-irritating, such as zinc stearate, and must on no account be used as a substitute for thorough drying.

The temperature of the bath may be gradually lowered as the baby gets older, and robust babies may be gradually accustomed to cold baths; in this way not only will the baby's heat-

regulating mechanism be trained to accommodate itself to changes of temperature, but the reduced temperature of the water will have a stimulating effect upon the infant's metabolism.

In addition to the daily bath the baby ought to be cleaned and powdered each time it has an evacuation of its bowels.

The habit of daily bathing ought to be continued throughout life.

(3) Attention to the Cord.—The cord should not be tied until it has ceased to pulsate, i.e., about ten minutes after birth, in order not to deprive the baby of a considerable amount of blood, unless there is some urgent reason to the contrary. The ligatures must be tightly applied, otherwise the placing of the child in its warm cot may sufficiently relax the umbilical arteries to cause secondary hæmorrhage. Owing to the absence of the circulation the stump of the cord begins to dry up and slough away. It falls off as a rule on about the tenth day. Great aseptic care is necessary in dealing with the cord in order to avoid infection, which may kill the baby either from tetanus or from erysipelas. It has been estimated by Keller that 20 per cent. of the neo-natal deaths in Berlin in 1904 and 1905 were due to infection from the umbilicus. Indeed, even when the greatest care is exercised infection sometimes occurs, and, according to Whitridge Williams, whenever a baby dies within the first fortnight without any obvious cause, post-mortem examination shows the cause to have been peritonitis due to cord infection.

After the cord has separated the umbilicus must be covered with dry sterile gauze and be kept free from moisture until it has been completely healed.

The practice of applying fuller s earth to the umbilical cord cannot be sufficiently condemned. It may introduce the tetanus bacillus.

Care of Genitals.—In the case of female infants the labia should be separated once a day and gently sponged with warm water (*without soap*) to remove all secretions. In cleaning such an infant after it has been soiled by a stool, the sponging should be done from before backwards to avoid the introduction of fæcal matter into the vagina. Male infants require very little attention to their genitals, except in cases of phimosis, for which circumcision is the most sensible treatment. Sometimes, in healthy children, there appears a hydrocele, either on one or both sides. This has no pathological significance and generally disappears spontaneously within four or six weeks. It requires no treatment.

Care of Mouth.—The greatest care must be taken in cleaning the infant's mouth for fear of rubbing away the delicate buccal epithelium, predisposing the mouth to infection. A little sterile cotton wound round the index finger should be very gently used, when necessary, for swabbing the mouth.

Care of the Ears.—The ear and the external auditory meatus must be well cleaned. The latter is effected by gently inserting a piece of fine thin linen into the meatus and turning it round upon itself. Linen is preferable to cotton wool, which may leave a few threads inside the ear.

Care of the Nose.—Immediately after birth the nostrils are cleared of mucus by squeezing to prevent its aspiration into the lungs, which may cause pneumonia.

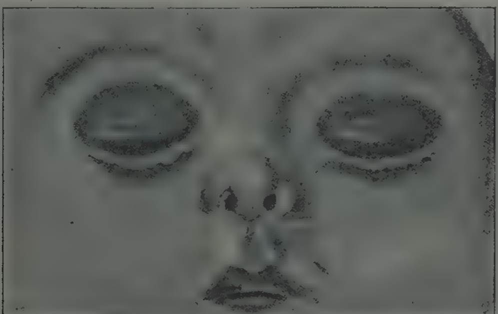

FIG. 81.—Ophthalmia neonatorum.

Care of the Eyes.—Figures collected by a Committee of the British Medical Association[1] show that in schools for the blind *ophthalmia neonatorum* (fig. 81) is responsible for the loss of sight of approximately 40 per cent. of the inmates. The cause of ophthalmia neonatorum is infection of the baby's eyes during its passage through the maternal passages in the process of birth. If the mother happens to have a vaginal discharge, some of it enters the baby's eyes and causes an inflammation which may either spread to the brain and kill the baby, or may merely destroy the transparent portion of the eyes, leading to permanent blindness. The organism responsible for this condition is, in the vast majority of the cases, the gonococcus, but other organisms in

[1] *Brit. Med. Journ.*, i, 1909, Editorial, p. 1138.

the vagina, such as the pneumococcus, Koch-Week's bacillus, streptococci, &c., may produce the condition, although in a much milder form.

Prevention.—(*a*) *Administrative.*—Every case of "purulent discharge from the eyes of an infant commencing within twenty-one days from the date of its birth" is now notifiable to the local Medical Officer of Health, either by the doctor or the midwife in attendance on the case, and in addition the midwife is obliged, under the rules of the Central Midwives' Board, to summon the aid of a doctor. Notification has been introduced as the result of the report of the Committee of the British Medical Association in 1909. Its object is: (1) to arouse public interest; (2) to secure early and skilled treatment; and (3) to afford reliable statistics of the incidence of the disease. The only objection that was urged against this measure was the slur that it might cast upon the parents' morality, but in view of the fact that the gonococcus is not the only germ responsible, the objection does not arise. Of late years provision has been made for the admission of such cases, together with the mothers, into special hospitals where expert treatment can be carried out.

The following figures (Table CVIII) give the number of cases of ophthalmia neonatorum that have been notified and that have died in England and Wales in the years 1920-23:—

TABLE CVIII.

	1920	1921	1922	1923
Notified ..	10,304 (10·8 per 1,000 births)	8,313 (9·8 per 1,000)	7,106 (9·0 per 1,000)	6,592 (8·7 per 1,000)
Died.. ..	46	40	25	16
Fatality rate	0·45 per cent.	0·48 per cent.	0·35 per cent.	0·24 per cent

From this we see that both the incidence as well as the fatality rate of this disease has been considerably diminished during the last few years.

(*b*) *Medical.*—Whenever there is the slightest suspicion of any maternal discharge, a few drops of a 1 per cent. solution of silver nitrate should be instilled into each of the baby's eyes immediately after birth. Since Credé introduced this method, the incidence of ophthalmia neonatorum in his own clinic fell from 10·8 per cent. to between 0·1 and 0·2 per cent., and on the Continent was reduced from 9 to 1 per cent. or less. In the

United States, according to Rosenau, notification and the Credé method diminished the frequency of ophthalmia neonatorum by 50 per cent. in three years (1911-14), and wiped it out altogether in 1919 and 1920. The silver nitrate should be washed away with normal saline solution.

In cases where no suspicion of gonorrhœa is present, the baby's eyes should be irrigated immediately the head is born with warm boracic acid solution instead of silver nitrate. The eyes should be washed from the nose outwards, and a separate piece of cotton wool should be used for each eye. In this way any risk of carrying infection from one eye to the other is avoided. The maternity nurse must also take precautions to avoid, by disinfecting her hands after attending to the mother, the carrying of infection from the mother's vaginal discharges to the baby's eyes.

We have seen, in Chapter II, p. 33, that Soranus, in the second century A.D., advocated irrigation of the eyes of the newly-born baby to prevent inflammation. In 1807 Gibson, of Manchester, recommended as prophylactics against the occurrence of ophthalmia neonatorum the following three principles: (1) Treat maternal leucorrhœa during pregnancy; (2) remove any vaginal discharge during labour; (3) cleanse baby's eyes immediately after birth with fluid. In 1830 Gottfried Eisenmann recommended the use of chlorine water for bathing the baby's eyes.

Care of the Breasts.—We have seen, in the previous chapter, that in both sexes during the first week the breasts may become engorged. The first manifestation is a little movable nodule under the skin of the breast, which increases in size and hardness in the course of a few days. It is important that no pressure or massage be applied with the object of emptying the milky fluid from the breasts. Such treatment will almost certainly result in an abscess. The breasts must be left alone and protected by means of cotton wool and a bandage from friction by the clothes. If this is done the engorgement invariably disappears.

The Stools.—For the first few days after birth the infant passes a dark green viscous substance called meconium. This consists of intestinal epithelial cells and mucus, and owes its colour to the presence of bile pigments. The meconium is sterile at birth, but within a few hours after birth bacteria gain access to it. After the third or fourth day meconium disappears, and is replaced by fæces which are light yellow in colour. Some authorities, e.g., De Lee, recommend the routine administration of ten drops of castor oil to the infant on the first day after birth, with the idea of clearing out all the meconium. This practice, according to some, helps to prevent colic. I believe such clearing

out to be unphysiological. The meconium supplies the necessary ballast for stimulating the intestines to perform their natural function during the first few days, and if it is expelled by castor oil the bowels remain empty until the flow of milk from the maternal breasts is sufficiently established to provide the infant with enough intestinal material to exercise its bowels. In this way the seed may be sown of subsequent constipation. If no meconium is passed within a short while after birth, the anus must be examined for occlusion.

The Urine.—The infant usually passes urine soon after birth. If no micturition has been noticed within twelve hours, it is necessary to examine the child to ascertain if there is any swelling in the lower part of the abdomen (distended bladder) before deciding that no urination has taken place unnoticed (e.g., in the bath). If the bladder is found to be distended, it shows that there must be some obstruction between the bladder and the urethral meatus, such as may occur in the case of a very tight prepuce or of congenital occlusion of the urethra. If when put into a hot bath (with the end of the penis out of the water to make evident any flow of urine) it still does not micturate, the probability is that the urethra is impervious, and surgical advice should be sought.

Training in Regular Habits.—The control of the bowels and bladder should be inculcated in the infant from the moment of birth. The baby should be systematically held out over a receptacle at regular intervals, so as to get it accustomed to perform its natural function at specified times. It is generally found that the exercise of a little patience and perseverance on the part of the nurse will accomplish a great deal with regard to the control of the bowels, although bladder control, especially during the night, is not generally acquired till the infant is many months old.

Suspended Animation or Asphyxia Neonatorum.—Sometimes infants are born in a condition of deficient or absent breathing, although the heart keeps on beating. Such a condition is spoken of as one of suspended animation or asphyxia, although the latter term, meaning as it does "pulselessness," is obviously a misnomer. The term asphyxia is, however, too firmly established in medical terminology to be replaced. Asphyxia may be of two kinds, viz:—

(*a*) *Blue Asphyxia.*—This kind, in which the baby looks blue, is not serious. It is due to some obstruction to the supply of oxygen caused by the presence of aspirated mucus or liquor amnii in the respiratory passages, or a lowering of the sensitiveness of the respiratory centre brought about by an excess of carbonic

acid gas in the blood. This latter may be caused by some interference with the placental circulation, such as premature separation of the placenta, excessive compression of the placenta due to prolonged strong uterine contractions, or compression of a prolapsed cord, &c.

The *treatment* of this condition is fairly easy and consists of clearing out the air passages either with a swab, or by suction through a rubber catheter passed into the trachea, slapping the baby's back and performing artificial respiration. The rubber catheter should contain a glass window to enable the operator to see the mucus before it reaches his mouth. The outlook is generally very good in such cases.

(*b*) *White Asphyxia.*—This kind, in which the baby looks white, and its muscles have lost all tone, as can be seen by the looseness with which the limbs hang from the body, is extremely serious. It has, however, nothing to do with obstructed respiration, and is caused by shock to the heart due to excessive cranial compression, which causes either slowing or acceleration of the rate, according as the vagus centre is stimulated or paralysed.

The *treatment* of this condition should therefore be directed to the heart rather than the respiratory system. The late Mr. Gordon Ley pointed out that in such cases it is the cardiac shock which has to be treated by keeping the infant very warm and at rest, as well as by stimulating its heart. This view is enthusiastically advocated by Professor McIlroy, who leaves such a baby at rest in a warmed cot and gently massages its heart under the blanket. She also gives injections of pituitrin or camphor hypodermically. In addition, any mucus in the trachea is sucked out by means of a rubber catheter, as in the case of the asphyxia.

Initial Loss of Weight (see Chapter X, p. 293).

The needs of the newly-born infant may be summarized as follows: (1) Warmth; (2) cleanliness; (3) soft loose clothing; (4) right kind of food; (5) sleep.

Identification of the Baby.—In Maternity Hospitals, unless some distinctive mark is given to every baby, mistaken identity is liable to arise. One such case has recently occurred in one of the Lying-in Hospitals in London, where a Christian baby was ritually circumcised, having been mistaken for a Jewish baby. To avoid such mistakes the following methods may be adopted:—

(1) Attach a label to the baby, with its name written upon it with waterproof ink.

(2) Take a finger-print, or preferably a foot-print (which is more easily obtained) of the baby.

(3) Write the baby's name on its back with weak silver-nitrate solution.

PART III

POST-NATAL HYGIENE

SECTION II

INFANCY STAGE

"It is more pleasant to the artist to paint than to have finished painting. . . . Henceforth it is the fruit of his art that he enjoyed while he was painting. In the case of our children their young manhood yields the most abundant fruits, but their infancy is sweetest."—SENECA, Epistle ix, 7.

CHAPTER XII

GENERAL NURSERY HYGIENE

"Observe due distances between his Meals,
Nor feed him when you find his Belly swells.
. . . . .
Wash him a Nights, ere you the Cradle make,
He'll sleep the sounder, and the sooner wake,
Stir him and toss him, for an infant's Sloth
Produces Rickets, and prevents his Growth.
. . . . .
Nor less to Dandle him and Dance Forbear,
Nor keep him in the House, but give him Air.
. . . . .
Show him the painted Skies, their rolling Fires,
Tell him Who made what he so much admires."

SCEVOLE DE STA. MARTHE
(*La Pædotrophia*,[1] 1584).

THE above lines, taken from a poem by a French doctor of the sixteenth century, give in rhyme the essentials of "Nursery Hygiene," viz., regularity of feeding, cleanliness, sleep, exercise, fresh air and sunlight.

The Nursery.—The ideal is to have a nursery flat consisting of day and night nurseries, a nurse's room, kitchen, bathroom and lavatory, but in many houses one room has to suffice. That room should be of good size and should provide about 1,000 cubic feet of space, after deducting the amount of space occupied by the furniture, for each child. The floor area should not be less than 100 sq. ft. per child. It should be on the top floor, to admit as much light as possible, and should if possible have two windows, facing in opposite directions, to allow for proper ventilation. If only one window is available, it should be facing south so as to admit the largest amount of sunshine. If a south aspect is unavailable, an east aspect is better than west, on account of the morning sun. If possible the window should have a balcony so that the baby can, in suitable weather, live outside. All windows should be provided with gauze screens to exclude flies and other

[1] Translated into English by the Abbot Quillet, in 1710.

insects, which are frequent sources of infection as well as annoyance, and should also be fitted with bars to obviate the danger of the child falling out. Hangings and pictures should as far as practicable be dispensed with.

The floor, which should be mopped twice a week, should be of hard wood or cork lino, and partially covered with small washable rugs. It should have no cracks. It should be cleaned with a vacuum cleaner by preference, but if this is not available the sweeping should be done when the baby is not in the room. To avoid raising the dust the floor and furniture should be wiped with a damp cloth. The baby should not return to the room for at least an hour after dusting or sweeping, to allow the dust to settle down. A young baby must be kept off the floor in cold weather on account of draughts from chinks, or from under the doors.

The nursery walls should, if possible, be of plaster with rounded corners where dust does not collect, and painted with washable paint of green colour (or, if papered, the paper should be free from poisonous dyes), and have an attractive animal frieze or pictures painted on the wall illustrating nursery rhymes or fairy tales. The designs of the nursery wall decorations should be so selected as to cultivate the child's taste in art and beauty.

Ventilation.—It is important that the nursery air should be kept as fresh and as sweet as possible, and hence it is essential to have a constant change of air from without. Cross ventilation, by means of open windows on opposite sides of the room, is best, the baby's cot being put out of the line of the air current ; but, if this is impossible, an open nursery window opposite a window in a passage or another room, or an open fireplace, will answer the same purpose.

In the winter, when it is not practicable to keep the windows wide open throughout the day and night, special ventilators may be used, or the windows may be opened slightly at the top and bottom. In addition the nursery air should be completely changed twice a day, by keeping the windows wide open for an hour at a time, during which time the baby should be taken into another room. Where the infant is bathed in the nursery, the airing should be done immediately after the bath. At no time may wet diapers be allowed to hang about in the nursery.

The Meaning of Fresh Air.—Till recently it was believed that the bad effects of expired air were due to its deficiency in oxygen and to the presence of impurities, such as excessive amounts of CO_2 and volatile organic poisons. This chemical theory, how-

ever, has now been abandoned, chiefly owing to the researches of Leonard Hill and other workers, who showed that it was the hot, stagnant air of an unventilated room, rather than its chemical composition, which renders it oppressive and harmful. The object of ventilation, therefore, is to keep the air cool and in motion. The fear of draughts is unfounded. If air comes into a room through a widely-opened window it enters at a slow rate and cannot do any harm. If the window is only slightly open, and the baby's ear or exposed feet happen to be in the line of the air current, then the air coming in as it does with a rush might produce local chilling on account of the too rapid evaporation from the part, with unpleasant consequences. The standard to be aimed at should be such as to correspond to that rate of cooling of the katathermometer which gives the most pleasant sensation.

The metabolic value of air in motion was recognised by Shakespeare, who makes one of his characters say, "The air is quick there and it pierces and sharpens the stomach."—(PERICLES, iv, 1).

With cradle, seen from within the room. "Designed to carry half a ton." Mattress and top cover on seen from outside.

FIG. 82.—Sunanair balcony, a device for exposing baby to fresh air in congested districts.

A baby may be safely allowed to lie in front of an open window if a screen is placed round the opposite side of the cot to impede the velocity of the incoming air. If the child's feet are kept warm and its head cool there is very little fear of its catching cold. The baby should preferably spend most of its time out of doors. Any mother who will pay a visit to the open-air hospitals and institutions for young children, where the inmates are constantly exposed to the open air and sunlight, would soon be convinced, by the wonderful results, of the baselessness of the fear of draughts. An electric fan is a very useful apparatus to keep the nursery air in motion when there is no screen.

Table CVI, p. 305, shows that a baby needs $400 \times 60 \times 24 = 576{,}000$ c.c. of air per day per kilo body weight. This is equivalent to about 720 grms. of air per kilo, or 72 per cent. of its body weight. As the daily amount of milk needed by the baby during its neonatal period is no more than about 15 per cent. of its body weight, we see that the infant needs about five times as much air by weight as food.

The Heating of the Nursery.—Central heating by steam, hot water, or hot air is useful, but in addition an open fire-place is a valuable adjunct, not only because it aids ventilation, but also because it can be used on cool days in the spring or autumn when the central heating is not in use. The chimney must never be allowed to be stopped, even when there is no fire in the grate. The temperature of the nursery should be about 70° F. by day and 65° F. at night, as measured with a thermometer. As the baby grows older the temperature can be gradually reduced, until at the end of the first year it is 45° F. Excessive heat in the nursery results in free perspiration with liability to catching cold, as well as in a lowered metabolism leading to pallor, indigestion and general debility, and even wasting. On the other hand, too low a temperature is also harmful, because babies cannot stand exposure to cold ; and secondly, the cold air, breathed in by the baby as it is, at the rate of about thirty times a minute, may irritate the delicate mucous membrane of its respiratory tract.

The humidity of the air should be about 50 to 53 per cent. (saturation being 100 per cent.). This can be secured by placing open dishes with water over the radiators, or a steam kettle over the coal fire.

The nursery fire-place ought to have a large guard, which should be big enough and high enough to prevent the child from touching or playing with the fire. This is a common-sense safeguard, specially legislated for in the Children Act, 1908. The Act adds that if no guard is provided, and a child under the age of 7 is burnt or scalded, the person in charge of the child may be liable to a fine of £10.

Lighting.—Gas lights are inadvisable in a nursery on account of their danger and the amount of oxygen they burn up, as well as the amount of irritating and offensive gases they produce on combustion. Electric lights are undoubtedly best, but they must be suitably shaded and subdued, so as not to irritate the baby's eyes. Where no electric light is available, a wax night light should be used.

FIG. 83.—Folding pram in use.

FIG. 84.—The same pram folded up.

The Perambulator.—The deep perambulators must be condemned on account of their stuffiness and lack of sunshine. The infant is almost buried deep down the vehicle, so that very little air and sunshine can reach it. Many pale babies can have their colour restored by raising them in their perambulators by means of thick mattresses to a level permitting them to come into contact with the invigorating, cool, pure atmospheric air, as well as with the health-giving rays of the sun. A folding perambulator useful for travelling is shown in fig. 83.

The nursery furniture should consist of the fewest possible articles, should be as plain, as strong, and as inexpensive as possible, but should be easy to clean and readily movable on castors. They should have sloping tops as well as rounded

FIG. 85.—Two-deck bed.

corners and edges, which not only make them more easy to clean, but cause as little damage as possible to any romping child who may happen to hit against them.

The following are the essential articles of furniture for the nursery or nurseries:—

The Cot.—This should be made either of hard wood or of iron painted with white enamel, and have sides which can be lowered for lifting the baby and for nursing purposes. It should not be placed in the draught. In cases of overcrowding two children may be accommodated in the same bed by using a two-deck bed (see fig. 85).

The Nurse's Bed.—If the nurse has no adjacent room for herself, her bed must, of course, be placed in the night nursery. The bed and cot should be so arranged that the cot is between the window and the nurse's bed. If there is more than one cot in the room, there should be a space of at least 5 ft. between them.

Bed Screen of simple washable material.

Cupboard with Drawers.

Table.

Nurse's Chair, which should be low and without arms.

Baby's Chair.—This should have a wide base, so that it cannot easily be turned over. No child should be put into a chair until it is about 8 or 10 months old, when it can sit up alone.

A Surgical Dressing Table, equipped with bath and clinical thermometers, the various articles for the baby's toilette and essential dressings, is a useful piece of furniture, as is also a *pair of scales* for regular periodic weighing of the baby.

Another useful article in the nursery is a refrigerator.

Nurse.—The selection of a nurse for a baby is a matter of very great importance. Such a person should preferably be of middle age, when she is no more so susceptible to the blandishments of the boys in khaki or boys in blue, must have a genuine love for babies, must be clean in body and in mind, must be strong, capable, energetic, and free from infection (e.g., septic teeth or tonsils, or chronic nasal catarrh). She must be intelligent, able to command discipline, and of a placid disposition. Her authority must be upheld by the mother.

The nurse must never be allowed to whip a child, and should never tell her elder charges terrifying ghost stories or tales of horrible disgusting animals, which may not only cause night terrors, but may sow the seed of a timid disposition in the future.

Sleep.—The younger the infant and the more rapid its rate of growth, especially that of the brain, the longer the number of hours of sleep that it requires, since it is during sleep that repair of waste takes place. During the first month it should sleep practically all the time, except when being fed and dressed. As the infant grows older the number of hours of sleep it requires gradually gets less, but even by the time it reaches the age of one year it should sleep about eighteen hours out of the twenty-four. It is, however, important that the baby should be so trained that it should sleep right through the night, from 6 p.m. to 6 a.m., without waking, except once at 10 p.m. for its last feed. Babies, after the age of about six months, should be

kept awake between 3 and 6 o'clock in the afternoon, in order to ensure good sleep throughout the night.

Every baby must be so trained as to fall asleep soon after it is put into its cot, without rocking. A baby accustomed to rocking will soon wake up if the movement is stopped.[1] If the baby cries it must within reason be allowed to do so until it falls asleep. To pick it up and carry it about the room will soon create a bad habit, from which it will be extremely difficult if not impossible to wean it. The baby should sleep on a mattress, which should be so inclined that the head lies a couple of inches higher than the lower part of the body, to prevent regurgitation of food in an infant prone to it, but it should have no pillow. It should be laid first on the right side, in which position the food easily finds its way into the duodenum, and its position should be changed at intervals in order to prevent deformity of the spine. It is hardly necessary to mention that under no circumstances should a baby sleep in the same bed as its mother or nurse, since, apart from the danger of overlying, this custom is in itself unhealthy for the child, because it allows the child to breathe the expired air of the other person. Also sleeping with the mother creates the temptation for frequent nursing. Under the Children Act, 1908, if an infant is suffocated whilst in bed with a grown-up person who was drunk when going to bed, that person is liable to a fine up to £100, or imprisonment with or without hard labour up to two years, or both. Those who cannot afford to buy a cot can improvise one for themselves out of a drawer or a banana crate.

While an infant should be trained to sleep in ordinary household noises, every care should be taken to prevent all sudden loud noises, such as banging of doors, when a baby is asleep, and no sleeping baby should be left alone with a cat or a dog, as accidents have occurred through the animal lying down on it and suffocating it. If it is impossible to keep an eye on the baby, a netting or similar device should be hung round the cot, so arranged that it can, when necessary, be drawn aside.

Amount of Sleep required.—During the first month the

[1] Rocking of babies is to be discouraged, because it creates a habit from which it is difficult to wean them. To say, however, that it stirs up the brain is a fallacy, because not only does the brain completely fill up the cavity of the skull, but the various intersecting membranes of dura mater make it utterly impossible to agitate it, in the same way as it is impossible to shake the water which fills a stoppered bottle. Hence the instinctive habit of women to sway a baby in their arms should not be discouraged.

child sleeps practically all day, except when it is fed. Between the second and sixth month it requires about twenty hours of sleep out of the twenty-four. At about six months a healthy baby generally sleeps twelve hours at a stretch at night, and an extra four hours during the day, two hours in the morning and two hours in the afternoon. The afternoon sleep should, however, be over by 3 o'clock, otherwise the baby might not sleep well during the night. At one year the day naps may be shortened to three hours, at eighteen months to two hours.

The twelve hours' sleep at night and an hour or two during the day are required by most children till the age of about six years. If the child is delicate or of a nervous temperament, it may require an extra hour of sleep during the day. In any case, even if the child cannot be induced to go to sleep during the day, it should be encouraged to rest for a while in the afternoon.

Before going to bed the child should empty its bladder and have an evacuation of the bowels. It should be completely undressed and be put into night clothing. None of the clothes worn during the day should be worn at night, but should be allowed to air and dry during the night to be ready for the next day.

TABLE CIX.

Hours of sleep	Intensity of current in milliamperes	
	9 months old boy (breast-fed)	3½ years old girl
1st	1,250	1,300
2nd	700	450
3rd	500	300
4th	450	200
5th	Woke up spontaneously for a feed	180
6th	—	150
7th	900	250
8th	650	350
9th	50	600
10th	Awoke	800
11th	—	350

It is fairly certain that children vary considerably with regard to the amount of sleep they require; a quiet, placid child requires less sleep than one that is active and excitable. According to Terman,[1] "undue emphasis has been placed by

[1] Terman, Lewis, *Journ. Educ. and Psych.*, 1913.

hygienists upon much sleeping. Sleep is only one of the needs of children, and should not be made the scapegoat for all the ills children are subject to."

Intensity of Sleep.—Czerny studied the depth of unconsciousness during sleep in children by ascertaining the intensity of an electrical induction shock required to wake them up after various intervals from the beginning of sleep. The preceding Table CIX illustrates his results.

From this table we see that the soundest sleep occurs during the first hour, that its depth decreases most rapidly during the second hour, and then gradually declines until about the sixth hour, when it reaches a minimum. After that sleep becomes sound again and reaches a second maximum, which, however, is less than the first during the beginning of sleep, during the tenth hour.

Results of Insufficient Sleep.—In the case of infants the results of insufficient sleep are irritability and crying. The baby also is liable to fall asleep at the beginning of a feed and will thus fail to take the proper amount of milk. This may lead to under-nutrition, or to feeding at irregular intervals with all the evil consequences thereof. In the case of older children there is nervousness, irritability of temper, lack of power of concentration, failure of memory, and all other signs of fatigue, such as inattention, drowsiness, &c. In addition there is loss of appetite, constipation and loss of weight, sallow appearance and even attacks of faintness.

The causes of sleeplessness in infants are :—

(1) Bad or irregular habits, such as being kept up late together with older persons, or being rocked to sleep.

(2) Insufficient or excessive clothing.

(3) Soiled diaper or cold feet.

(4) Indigestion due to improper or irregular feeding.

(5) Hunger.

(6) Thirst.

(7) Dentition, especially in rickety babies.

(8) Badly ventilated rooms, especially in infants suffering from nasal obstruction.

The remedy lies in discovering the cause and removing it.

In older children the causes are similar to those in the case of infants, with the addition of mental fatigue due to overpressure at school. A custom prevailing at the open-air nursery schools is to put aside two hours in the early afternoon for the

purpose of sleep. This is a custom which might be copied in the case of more weakly children in the elementary schools.

The Influence of "Summer-time" on Children's Sleep.—With the commencement of "summer time" there is of course loss of an hour's sleep. This may lead to a state of fatigue in a school child, and some latitude should be allowed at school with regard to the ordinary lessons or home work at the commencement of "summer-time." Perhaps the best way of overcoming the difficulty is to start school an hour later on the day on which "summer-time" commences.

The Board of Education published a statistical report of the views expressed by the various Education Authorities throughout England and Wales, in 1921, on the question of "summer-time." Of about 300 replies received, representing a total population of 5,000,000 school children, the proportion of those in favour to those against "summer-time" was approximately 2 to 1. But even those that were against it on the score of deprivation of sleep were of opinion that proper parental control would do away with this objection to the Act.

Sunlight.—The importance of sunlight in the hygiene of infancy is dealt with at length in Chapter XX. Here it is only necessary to mention that no opportunity should be neglected for keeping the child in the open when the sun is out provided the head and back of the neck are protected by a large hat when the sun is very hot. In the winter every ray of sunshine must be utilised. It must be remembered that a closed window, though it will admit the light and heat rays, will exclude the beneficent ultra-violet rays, unless special glass, such as vita-glass, is employed.

Three things must be remembered in the case of infants exposed to the sun:—

(1) Exposure to the direct rays of the sun on a hot summer day, when the humidity of the air approaches to the point of saturation so that perspiration is checked, may tend to sunstroke, as the result of the accumulation of body-heat.

(2) The danger of causing severe dermatitis, especially in infants that do not pigment well. Such children require gradual exposure until they become accustomed and inured to the sun rays.

(3) The sun rays are much more powerful at high elevations than at sea-levels.

LITERATURE.

Feldman, W. M. "A Manual of Nursery Hygiene," London, 1912.

CHAPTER XIII

THE NUTRITION OF THE INFANT AND THE CHILD

"Mute and still, by night and by day, labour goes on in the workshops of life. Here an animal grows, there a plant, and the wonder of it all is not the less in the smallest being than in the largest."—RUBNER.

A PROPER diet for both adults and children must contain the following constituents: protein, fat, carbohydrates, salts, water and vitamins. These constituents are needed by a growing child in quantities different from those required by an adult. Also, in view of the immature development of its digestive apparatus, an infant requires these food substances in forms suitable for its digestive capacity. In addition, a growing child requires proportionately more food to allow for the formation of new tissue.

Food Requirements of an Infant.—An estimate of the *amount* of food required by an infant must be based upon the following considerations:—

(*a*) *Total caloric requirements*, as estimated from the amount of heat lost by the infant during the day, together with the amount of energy required for the purpose of growth. Food, of course, is the fuel of the body, which on being burnt up by the oxygen inspired from the air, gives rise to body-heat. This total is represented by the algebraic sum of:—

(i) *The amount of basal metabolism*, viz., the amount of heat produced by a fasting infant (i.e., one in whom there is no energy spent in the process of digestion) in a state of muscular rest. This represents the energy production of the body apart from functional activity, other than that of the circulatory and respiratory systems. As the first condition is in the case of infants incompatible with the second, and as the energy of digestion is very slight in the case of babies, it is sufficient for practical purposes to define basal metabolism in infants as the amount of heat produced by it during deep sleep, even soon after a

feed. This is fairly constant for babies of the same weight, and represents *the minimum maintenance ration.*

(ii) *The amount of heat used up by muscular activity.* This, of course, is a variable factor depending upon age, &c.

(iii) *The growth quotient*, i.e., the extra amount of food required for growth. This diminishes with the age of the infant. The idea of the growth quotient was recognised by Hippocrates who, in his "Aphorisms" (i, 14), says: "The growing organism has the most innate heat, and therefore requires most nourishment."

(iv) *The amount of food lost in the excreta*, and therefore not utilised by the body for metabolic purposes.

Calories.—Food substances burned up in the body produce the heat which is necessary for life. The exact amount of heat produced by each of the different food substances can be calculated in terms of calories or heat units, and knowing the amount of heat lost by the body in the course of twenty-four hours, the exact amount of food required to supply the necessary calories can be ascertained. A *calorie* is the amount of heat necessary to raise a kilogramme of water from 0° to 1° C. The calorie employed by physicists is $\frac{1}{1000}$ that employed by physiologists, and is the amount of heat necessary to raise 1 grm. of water from 0° to 1° C.

The heat energy of the food is in part transformed inside the organism into dynamic energy, viz., work and movement. The calorie is equivalent to 426 kg. metres (i.e., the energy required to raise 426 kg. one metre high), but, owing to the efficiency of the human machine being no more than about 20 per cent., the human body can only produce about 85 kg. metres of work from each calorie.

(*b*) The distribution of the total calorie requirements between the various essential food ingredients: protein, fat, carbohydrate.

(*c*) The chemical character of the food ingredients, especially that of protein, from the point of view of its amino-acid constitution.

(*d*) The amount of salts.

(*e*) The amount of water.

(*f*) The presence of vitamins, especially vitamins A and C.

(*g*) Lastly, the digestibility of the food must also be considered.

Caloric Requirements.

(i) *Basal Metabolism.*—The following figures (Table CX) giving the basal metabolic rates of infants from birth onwards, are based upon the calorimetric researches of Benedict and Talbot, as well as of Murlin and Hoobler :—

TABLE CX.

Age of child	Basal metabolism in calories — Per kg. per day
0 to 12 hours	38
12 hours to 2 weeks	42
2 to 4 months	57
6 to 12 ,,	59
1 year	58
2 years	52
3 ,,	50
4 ,,	47
6 ,,	44
10 ,,	38
15 ,,	30

(ii) *Extra Heat used up by Muscular Activity.*—This is a variable factor, depending upon the amount of activity, crying, &c. Experiments have shown that on the average muscular activity accounts for another 50 per cent. of the basal metabolism, but excessive crying may raise the metabolism by as much as 200 per cent.

(iii) *The Growth Quotient.*—If the infant is to gain normally, i.e., at the rate of about 25 grm. a day (for an infant weighing 5 kg.), one must add the caloric value of 25 grm. of the infant's body tissue, as calculated from the average chemical composition of a baby. This works out at the rate of 1·87 calories per gramme, or 47 calories in an infant weighing 5 kg. for a total daily gain of 25 grm., i.e., at the rate of about 9 calories per kilogramme of body-weight = 15 per cent. of basal metabolism.

(iv) *The Loss by Excretion.*—This, according to recent work by Holt, Courtney and Fales, amounts to about 10 per cent. of the food intake in the case of breast-fed babies, whilst in the case of artificially-fed babies an extra allowance of 15 per cent. must be made.

If we add up the various factors we find that the total caloric requirements of an average baby, growing normally, are as follows :—

Basal metabolism ..	60 calories per kilogramme
Muscular activity (50 per cent. of basal) ..	30 ,, ,, ,,
Growth quotient (15 per cent. of basal) ..	9 ,, ,, ,,
Loss in excreta (10 per cent. of total intake) ..	11 ,, ,, ,,
Total	110

We see, therefore, that the total caloric requirement is 110 calories per kilogramme, or about 50 calories per pound. This estimate is slightly less than the amount necessary during the first few months when growth is most rapid, and slightly more than that required during the subsequent months of infancy. Also, artificially-fed babies require slightly more on account of the greater loss through the excreta (especially fæces) as well as the greater amount of energy used up by such babies in the process of digestion, &c. During the first few days of life the total caloric requirements are not more than about 60 per kilogramme, or about 28 calories per pound body-weight. Table CXI (after Holt) gives the total caloric requirements at different ages:—

TABLE CXI.

Age	Caloric requirement	Amount of milk	
		In oz. per lb.	In proportion to weight of baby
1st week	60 per kg. = 28 per lb.	1·4 oz.	about 10 per cent.
2 to 3 weeks.. ..	100 ,, ,, = 45 ,,	2·25 ,,	,, 14 ,, ,,
6 weeks	120 ,, ,, = 55 ,,	2·75 ,,	,, 17 ,, ,,
1 year	100 ,, ,, = 45 ,,	2·25 ,,	,, 14 ,, ,,
6 to 16 years (boys)	80 ,, ,, = 37 ,,		
Adult	44 ,, ,, = 20 ,,		

In the case of girls the same caloric requirements hold good up to about 6 years. After that, their requirements are slightly less. The determination of caloric requirements from the surface area is probably more accurate than from the body-weight, but it is somewhat more complicated, and on account of the difference between the two methods in the case of persons differing slightly in weight being very slight, the body-weight is a much more convenient unit to use in practice.

The reason why weight is as good a unit as surface for estimating the relative caloric requirements of children differing very slightly in weight, but not those the difference between whose weight is considerable, is a mathematical one, depending on the binomial theorem, but it will be readily understood from a concrete example.

Supposing we have two children whose weights are 5·5 and 5 kg. respectively. Their weight ratio is obviously 1·1 : 1, whilst the ratio between their surfaces (using the formula $S = KW^{\frac{2}{3}}$) is 3116 : 2924 = 1·06 : 1, so that their surfaces are practically in the same ratio as their weights.

If, however, the children whose caloric requirements are in question have weights of, say, 10 and 5 kg. respectively, then their weight ratio is 2 : 1, whilst their surface ratio is 4920 : 2924 = 1·61 : 1. In other words,

whilst in the first case the difference between the two ratios is only about 4 per cent., the difference in the second case is 20 per cent. Hence if, as it is believed, the heat loss is proportional to the surface area of the body, there is very little difference in the caloric requirements of the first pair of children, whether the calculation is made by weight or by surface, whilst in the second pair of children there would be a discrepancy of 20 per cent. between the two estimates.

It must be repeated that the above requirements represent averages and need not necessarily hold good for any particular child. Under-weight babies require more than the average, and babies weighing more than the normal for their age require less. Nervous, irritable and crying children require a little more than quiet and placid ones.

Again, during the summer less calories are required than during the winter, although, on account of greater loss of water by perspiration, the amount of water must certainly not be diminished (see p. 432), and the amount of the baby's clothing makes a considerable difference; the more the clothes the less the caloric requirements. A very active baby requires more than a quiet one. Finally, bottle babies require more than breast-fed ones, because of the greater amount of energy used up in the process of digestion as well as the greater loss through the excreta.

Distribution of Total Calories between the Various Food Ingredients.

Protein.—Observations on normal breast-fed babies show that they take with the milk about 1·5 grm. protein a day for every kilogramme of body-weight, i.e., about 0·15 per cent. of body-weight. Artificially-fed infants require considerably more than that amount on account of the greater amount of nitrogen waste (in the urine and fæces) in such babies, as well as the deficiency of the essential amino-acids in the protein ingested.

Since protein is the only food ingredient which contains nitrogen, an essential element in the formation of protoplasm, it is obvious that **protein is a substance which cannot be replaced in the diet by any other ingredient.** The adult requires protein to replace wear and tear of tissue (i.e. the nitrogen excreted in the urine and fæces, &c.), but the growing organism, as we have seen, further requires an extra amount for growth. During the process of protein digestion and metabolism, however, it also produces **heat of combustion,** which is equivalent to **4 calories per gramme** of protein **or 116 calories per ounce.** In this way,

therefore, protein is not only a tissue builder but a heat producer. The protein contribution to caloric requirements is therefore $1 \cdot 5 \times 4 = 6$ calories, i.e., about 6 per cent. of the total daily requirement.

It is to be noted, however, that for the purpose of tissue building not all proteins are alike, and the infant requires those kinds of protein whose structure is such as to yield, on digestion, all the various amino-acids or so-called building stones required for maintenance as well as growth. Such proteins are the caseinogen and lactalbumin of milk (see further p. 363 *et seq.*).

Proteins do not normally enter the blood as such, but are first broken up by the various digestive juices into their separate amino-acids.

Protein digestion begins in the stomach, where it is acted upon by pepsin, rennin and HCl, and continues in the intestines under the action of trypsin and erepsin. All these ferments are present in the alimentary tract of the infant at birth, although the amounts are then very small, and increase as the infant grows. The pepsin and rennin convert the protein into peptone, and the trypsin and erepsin split the peptone up in the intestines into amino-acids, in which form they are probably entirely absorbed.

Relative Proportions of Nitrogen absorbed from Different Proteins.—The prevalent teaching is to the effect that artificial feeding is inferior to breast feeding, on account of the relative indigestibility of cow's casein. This belief, however, seems to be erroneous, because accurate experiments by Keller and Orgler[1] have shown that the amount of fæcal nitrogen is not only not more but actually less in babies fed on modified cow's milk than on the breast, as the following example will suffice to show :—

Table CXII

	Nitrogen intake	Fæcal nitrogen
Breast-fed	1·51	0·29
Cow's milk	2·05	0·27

Here the intake of nitrogen was greater in the case of cow's milk, and yet the amount of fæcal nitrogen was less. The

[1] A. Orgler, "Der Stickstoffwechsel des Säuglings," *Ergebn. d. inn. Med. u. Kinderheilk.*, ii (1908).

proportion absorbed being 87 per cent. in the case of cow's milk as against 81 per cent. in the case of human milk.

One must, however, distinguish between the amount of nitrogen absorbed and that retained in the system, for the greatest amount of nitrogen is excreted in the urine. The experiments of Keller[1] and others show that when the same infant is fed on the breast and on cow's milk, although the amount of fæcal nitrogen is less when the infant is artificially fed, the urinary nitrogen is so much higher that the amount of nitrogen retained in the system is higher when the baby is breast-fed. This is illustrated by the following experiment :—

TABLE CXIII.

Food in grm.	Nitrogen in grm.			Nitrogen retention	
	Food	Urine	Fæces	Absolute in grm.	Per cent. of intake
850 breast milk	1·87	0·24	0·79	0·84	45
320 cow's milk + 480 water + 24 lactose	2·22	1·33	0·14	0·76	34

Other evidence, showing that cow's caseinogen is not of an inferior digestibility to that of the human variety, is afforded by the ingenious "**whey-exchange**" experiments of Ludwig Meyer.[2] This observer interchanged the whey and curd of human and cow's milk, and fed a number of normal infants as well as a number of those recovering from enteritis. He found that the weight curves as well as the stools of those fed on human whey, whether containing cow's or human caseinogen, were invariably better than those fed on cow's whey, even if it was combined with human caseinogen. Hence any bad effects which may result from cow's milk are not due to caseinogen but to the whey.

Moreover, Meyer obtained similar results when the lactalbumin was removed from the whey. Hence, according to him, it is probably the milk salts, which are three and a half times as abundant in cow's milk, that by their irritation of the infant's intestinal mucosa are responsible for the bad effects of cow's milk.

It must, however, be remarked that in this hypothesis it is very difficult to explain the very beneficial effects that are often observed after feeding with whey in cases of digestive disturbances of infancy.

1 *Arch. f. Kinderheilk.*, xxix, 1900.
2 *Monatsschr. f. Kinderheilk.*, v, 1906.

Influence of Carbohydrates and Fats on Nitrogen Retention.—*Carbohydrates.*—Excessive intake of sugar will cause increased intestinal peristalsis with consequent increased loss of nitrogen in the fæces. On the other hand, the extra carbohydrate will simultaneously bring about a reduction in urinary nitrogen to such an extent as to more than compensate for the increased loss in the stools. The net result is that more nitrogen is retained in the body, so that **carbohydrate acts as a protein sparer.** This is well seen from the following table, given by Keller,[1] in the case of a six months' old infant :—

TABLE CXIV.

Food	Nitrogen in food	Nitrogen in urine	Nitrogen in fæces	Nitrogen retained (grm.)	Nitrogen retained per cent. of intake
½ cow's milk + 75 grm. maltose	2·04	1·323	0·239	0·478	23·5
Whole milk	3·83	3·189	0·188	0·438	11·9
Whole milk + 75 grm. maltose	3·83	2·744	0 347	0·739	19·3

This table shows that in each case when maltose was added to the milk, although the fæcal nitrogen was increased, the *retention* of nitrogen in the body also increased because the urine nitrogen was greatly diminished. The explanation of such a striking phenomenon, offered by Talbot and Hill, is that in the presence of extra carbohydrates the fæcal bacteria act upon them instead of upon the protein, of which a greater amount is consequently left for absorption. It is difficult to see, however, how such action could increase the amount of fæcal nitrogen, except on the supposition that the increased peristalsis caused more nitrogen to be discharged from the intestinal mucosa.

Fat has no influence upon the proportion of fæcal nitrogen. This is shown in the following experiment by Freund[2] :—

TABLE CXV.

Food	Nitrogen in food	Nitrogen in fæces	
		Total	Per cent. of intake
Buttermilk	3·405	0·379	11·1
Buttermilk + Butter	0·114	0·338	10·9

[1] A. Keller, "Ueber den Einfluss der Zufuhr von Kohlenhydraten auf den Erweisszerfall," &c., *Zentralbl. f. inn. Med.*, xx, 1899.

[2] Walter Freund, "Zur Wirkung der Fettdarreich auf den Säuglingsstoffwechsel," *Jahrb. f. Kinderheilk.*, lxi, 1905.

Hence we learn that in certain cases of defective growth it is advisable to replace fat by an isodynamic amount (i.e., an amount giving the same quantity of calories) *of carbohydrate.*

From all these experiments we see that in the ordinary way the amount of fæcal nitrogen is approximately 10 per cent. of the amount ingested. It is to be remembered, however, that as fæcal nitrogen contains also a certain amount of nitrogen coming from the intestinal secretions, the amount of nitrogen absorbed and metabolised (though not of course retained, a large quantity being excreted in the urine) is more than 90 per cent. of the amount ingested.

Nitrogen in Fæcal Curds.—The curds which sometimes are found in infants' stools are not necessarily an indication of disease. The small soft curds contain a percentage of nitrogen which is normal for fæces and a high percentage of fat. The large hard curds have a high percentage of nitrogen and a small percentage of fat. The nitrogen comes from the casein.

Fats.—The importance of fat to the infant can be realised from the fact that 12 to 13 per cent. of the normal baby's body-weight is fat, while atrophic infants have only 0·2 to 2 per cent. of fat. Hence it is possible for a baby to continue to grow in length (on account of the deposition of protoplasm), but at the same time to go on losing in weight on account of the burning up of fat. Fat when burned up yields 9·3 calories per gramme (or 264 calories per ounce) and hence is a great heat-producing substance. On the other hand, it is quite certain that fat is not absorbed as such through the intestine, but is split up first into glycerine and fatty acids, by the bile and pancreatic fat-splitting ferment which is present at birth. The fatty acids combine with alkaline carbonates to form soaps, in which form they are absorbed. Such being the case, and also from the fact that fat can be formed in the body both from protein and carbohydrates, it is not quite clear what the real function of fat in the diet is. That it is, however, of great importance is obvious from the high fat content of breast-milk (3·5 per cent. as against 1·5 per cent. of protein). Its high calorific value makes it of course a great source of heat to the infant. It is equally known that fats contain the fat-soluble vitamins A and D, which are essential for growth and the prevention of rickets. It is also probable that fats aid in the metabolism and absorption of minerals, especially calcium, ince, as we have seen, fats after being split up combine with these alkalis, and are absorbed as soaps into the blood. Taking

the amount of fat ingested by the infant from the breast-milk in the course of a day as the basis, we can say that *a baby requires 4 grm. of fat per kilogramme body-weight per day* (*i.e.*, 0·4 *per cent. of its body-weight*).

Fat in the Infant's Fæces.—In the same way as fæcal nitrogen comes both from the food as well as from intestinal secretion, so does fæcal fat come from both these sources, as is shown by the fact that when an infant is fed on food containing no fat, there is still an amount of fat in the fæces equal to 0·82 per cent. of the fæcal weight.[1]

Amount of Fat utilised in the Body out of the Fat Ingested.—Recent exact analyses by Holt and his co-workers[2] have shown that in the case of normal breast-fed infants the proportion of fat utilised varies between 90 per cent. and 99 per cent. with an average of 95 per cent., whilst in the case of bottle babies, those that were normal utilised over 91 per cent. of the fat ingested. In the case of bottle babies suffering from some digestive disturbance, the amount of fat absorbed is much less, and the amount of fat in the fæces is correspondingly increased. Thus, in severe diarrhœa the proportion of fat retained may be as low as 58·5 per cent. the amount in the stools being 41·5 per cent. of that taken in the food. As regards the distribution of the fat in the stools between neutral fat (i.e., unsplit fat), soaps and free fatty acids, the highest proportion neutral (unsplit) fat occurs in cases of diarrhœa, since in such conditions there is less chance for the fat to be split up.

Factors Influencing the Utilisation of Fat.

(1) *Calcium.*—It has been urged by many observers that high calcium intake, as occurs in feeding with cow's milk in which the proportion of calcium is high, results in the formation of insoluble calcium soaps, in consequence of the combination of calcium with the fatty acids, and thus not only hinders the absorption of fats, but also gives rise to a loss of calcium. The researches of Holt, Courtney and Fales,[3] as well as those of Fife and Veeder[4] have shown, however, that neither in normal nor in atrophic infants is there any relation between the amount of fat and calcium excreted in the stools.

[1] A. Keller, "Phosphor und Stickstoff im Säuglingsorganismus." *Arch. f. Kinderheilk.*, xxix, 1900.

[2] Holt, Courtney and Fales, "Fat Metabolism in Infants and Young Children," *Am. Journ. Dis. Ch.*, 1919-1920.

[3] *Loc. cit.*, 1920. [4] *Ibid.* ii, 1911.

(2) *The Nature of the Fat.*—The work of Arnschink, in 1890, showed that the percentage of fat lost in the fæces, was in direct ratio to its melting point. Thus stearin, with a melting point of 60° C. was excreted in the fæces to the extent of about 90 per cent., whilst olive oil, which is fluid at ordinary temperatures, was excreted to the amount of 2·3 per cent. of the quantity ingested.

(3) *Carbohydrates and other Foods.*—These have little effect upon the utilisation of fat.

(4) *Alimentary Secretions.*—Physiological considerations would lead one to expect that any pathological condition interfering with the secretion of either pancreatic juice or bile would naturally affect the utilisation of fat. This is found to be the case. In cases of congenital absence of bile-ducts, studied by Koplik and Crohn, and Niemann, not only was the total fat excretion enormously raised, but the distribution of fats was also greatly changed, the amount of unsplit fat being raised. Thus in Koplik and Crohn's case, in a baby 10 weeks old the utilisation of fat was only 48·4 per cent., and the percentage of neutral fat in the fæces was 77·4 per cent. as against 20·2 per cent. in breast-fed, 9·4 per cent. in normal bottle-fed babies. Similar results were found by Finizio in a case of acute pancreatitis in a baby 11 months old. In that case neutral fat formed 82 per cent. of the fæcal fat.

Fat Intolerance.—While the fat in human milk causes very little if any trouble, the fat of cow's milk is probably the most difficult to digest and absorb. This is not due to the greater percentage of oleic acid in human milk, nor to the smaller size of the fat globules in it. It is almost certainly due to the medium in which the fat is given, namely, the whey, since protein milk or casein milk (or Eisweissmilch), which contains very little whey and whey salts, and a practically normal amount of fat, is the easiest nourishment that a delicate baby can take. L. F. Meyer,[1] in 1914, prepared "interchanged whey mixtures," consisting of human whey and cow's fat, as well as of cow's whey and human fat, and he found that babies fed on the first mixture thrived, whilst those fed on the second failed to gain in weight and began to suffer from digestive troubles. Lichenstein and Lindberg[2] repeated these experiments on a large number of infants and were unable to confirm Meyer's conclusions.

The most important symptom of cow's milk fat intolerance

[1] Quoted by Brennermann, in Abt's, "Pediatrics," vol. ii, p. 639.
[2] *Jahrb. f. Kinderheilk.*, lxxxix, 1919.

is vomiting, which cannot of course be due to its indigestibility, since fat is not digested in the stomach, but is due to the longer delay of cow's milk fat in the stomach, as compared with human milk fat.

Carbohydrates.—Carbohydrates can only enter the circulation as monosaccharides or hexoses, and hence, when taken in any other form, are first acted upon by the various ferments to be converted into hexoses. This action is started by the diastase in the saliva, and this action is continued by the lactase, invertin, and maltase in the intestine. All these ferments are present at birth and increase in amount with age. Carbohydrate has a calorific value of 4 calories per gramme, or 116 per ounce (the same as protein), and containing as it does exactly the same elements as fat, is to a certain extent interchangeable with it. Both supply heat and energy, although fat is more than twice as heat-producing (9 per gramme, or 263 per ounce) as carbohydrate, and neither of them can replace protein. Carbohydrate, as we have seen, can spare protein to a considerable extent. Also, carbohydrate can *for a short while* replace fat, but fat cannot replace carbohydrate, since the complete combustion of fat cannot take place in the absence of carbohydrate. **Without carbohydrate, fat burns incompletely, producing ketone bodies, which are responsible for ketosis or acidosis.**

The carbohydrate of milk is *lactose* or *milk sugar*, which is a disaccharide. It is present in practically constant proportions, viz., 7 per cent. in human and 5 per cent. in bovine milk, but whilst in human milk the 7 per cent. of lactose probably never causes any digestive disturbance, such an amount given in cow's milk would soon upset the infant by causing fermentative changes resulting in diarrhœa with loose, acid, frothy, irritating, liquid stools. Here again, therefore, as in the case of protein and fat, it is the vehicle in which the lactose is conveyed rather than the lactose itself which determines the tolerance of the infant towards this substance.

Cane sugar (*saccharose*), which is also a disaccharide, has the same nutritive value, the same calorific value and the same tolerance limit as lactose, and, being much cheaper than lactose, can be economically and safely used in the place of the latter, except in wasting babies, when it is not so good as the other sugars for reasons stated below.

Maltose and dextrine compounds are tolerated by babies in greater amounts than the other sugars, and hence may be used instead of other sugars in cases of diarrhœa due to sugar intolerance,

or in cases of fat intolerance, when it is needed to replace an isodynamic quantity of fat.

Milk sugar is converted by the specific ferments into dextrose and galactose, cane sugar into dextrose and lævulose, maltose is split up into two molecules of dextrose, and dextrine is first converted into maltose and then into dextrose. As already stated, it is only in the forms of monosaccharides, dextrose, lævulose, and galactose, that sugars can be absorbed and utilised by the blood. Dextrose is the normal carbohydrate of blood, and of all the monosaccharides is the one most easily absorbed. It is also least easily fermented in the intestines. Lævulose and galactose, which constitute 50 per cent. of the digested cane sugar, and milk sugar, are more easily fermentable, are less readily absorbed into the blood, and must first be stored as glycogen in the liver before they are transformed into dextrose and utilised by the body. This explains the advantage of dextrine-maltose preparations over other sugars in wasting babies.

Starch (as given, for instance, in barley water, Ridge's food, Allenbury's food No. 3, Robb's biscuits, Robinson's patent groats, &c.) is a poly-saccharide. It cannot, of course, be absorbed as such, and must first be converted into dextrine, then into maltose, and finally into dextrose. The amylolytic ferments of the salivary glands and the pancreas are present at birth, and therefore even the youngest baby can deal with moderate amounts of starch. Indeed, the gradual conversion of the starch into absorbable and utilisable sugar produces less dextrose at any time to undergo fermentation, and is therefore used with advantage in fermentative disorders with diarrhœa. The blood-sugar curve of a baby given starch by the mouth is of the same type as that given by a feed of sugar. On the other hand, in very feeble babies there may not be enough amylolytic ferment to enable them to digest starch.

The relation between protein carbohydrate and fat may be thus summarised:—

(α) The protein molecule contains a carbodydrate moiety, so that, when metabolised, protein produces 60 per cent. of its weight of carbohydrate.

(β) Protein cannot, on account of its nitrogen content, ever be replaced by fat or carbohydrate.

(γ) Fat burns in the flame of carbohydrate (1 grm. of carbohydrate, whether taken as such or produced in the body from protein, burns up 3 grms. of fat), and unless therefore there is a sufficient amount of carbohydrate taken, incomplete fat combustion may occur and ketosis or acidosis will result.

(δ) Carbohydrate can for a certain time replace fat, but owing to its water absorption, and also to the fact that animal fats contain vitamins and help in the metabolism of calcium, such substitution must not be prolonged.

Mineral Salts.—The various mineral salts present in milk are necessary not only for growth of the skeleton but also for the vital processes of the body as a whole, since the child's body contains 2 to 3 per cent. of salts. This is proved by the fact that not only will an animal cease to grow, or even to maintain its weight, if minerals are eliminated from its diet, although protein, fat, carbohydrate, water as well as vitamins are supplied, but the amounts of these minerals in the milks of various animals are proportional to the rates of growth of the young of those animals (see p. 402). Indeed, this is what we would expect, since any nitrogen retained from protein must be built up again into protoplasm, and to accomplish this there must be salts available. About 0·3 grm. of ash is required for each 1 grm. of protein. The exact rôle, however, played by any one of these salts in the physiology and hygiene of infancy is not at all well understood, and until exact feeding experiments in animals are carried out, in which the effects of the exclusion of one after another of the different salts for long periods are observed, the specific significance of each of them will never be known. Such experiments have not yet been conducted on any elaborate scale. We do know, however, that calcium, magnesium and phosphorus are essential for skeletal growth, and that in rickets the percentage of these in the blood is diminished; that chlorides are necessary for the formation of gastric juice; that calcium and sodium have an influence on muscular irritability, the former inhibiting it and the latter increasing it. Phosphorus is also used for the growth of the nervous system. As regards the metabolism of salts, it is known that their retention and absorption into the blood is favoured by fats and in a less degree by carbohydrate. But whilst the mineral salts play a most important, though not clearly understood, part in the metabolism of infancy, they contribute nothing towards the caloric requirements of the body.

The quantitative requirements of the various minerals by the body have, as I stated, not been studied so intensively as the calorific requirements of the body. It is only in the case of calcium and phosphorus that one has any idea at all as regards the probable minimal needs. It is said that the adult requires 0·006

grm. of calcium and 0·013 grm. of phosphorus per kilogramme body-weight. The requirements of the growing infant, when bones and brain are growing rapidly, must be more, but the exact minimum is not known, apart from the amount ingested by the infant with the breast-milk. According to A. Gautier, an infant requires about 0·5 grm. calcium phosphate a day for the purpose of building its body tissues. We know, however, that the excess of salts in cow's milk does not cause any perceptible harm to the infant. According to Holt, the salt metabolism in the case of breast-fed and bottle babies may be represented as follows :—

TABLE CXVI.

Food	Intake of salts	Excreted in		Absorbed	Retained	Per cent. retained
		Urine	Fæces			
Breast milk ..	1·29 grm.	0·44	0·23	1·06	0·62	49·5
Cow's milk ..	5·78 ,,	2·63	1·90	2·88	1·25	14·8

Calcium cannot be absorbed in the absence of fat, and observations have shown that, strange as it may appear, the best absorption takes place when the proportion of calcium to fat is that present in cow's milk, viz., 1 : 20.

The requirement of iron by an adult is 0·008 grm. per kilogramme. The infant apparently requires less (judging from the iron content of milk), because the new-born baby stores a large amount of iron in the liver. Nevertheless, infants fed on the breast alone for too long are apt to suffer from anæmia, and hence iron-containing foods such as beef-juice, yolk of egg, &c., should be added to the baby's dietary after the seventh or eighth month. Krasnogorsky[1] found that although mother's milk contains less iron than goat's milk (about 3 and 1·75 mg. Fe_2O_3 per litre respectively), more iron is absorbed and retained by the infant from the former than from the latter, in the proportion of 88 per cent. to 25 per cent. Cow's milk contains less iron than mother's milk (about 0·5 mg. per litre).

The following table is given by Tobler and Noll[2] of the retention of the various mineral substances by an infant two and a half months old in order to form 100 grm. of body tissue :—

[1] N. Krasnogorsky, "Ueber die Ausnützung des Eisens bei Säuflingen," *Jahrb. f. Kinderheilk.*, xliv., 1906.

[2] Tobler and Noll, "Zur Kentniss des Mineralstoffwechsel bei Gesunden Brustkind," *Monatschr. f. Kinderheilk.*, ix., 1909.

TABLE CXVII.

K_2O	Na_2O	CaO	P_2O_5
0·69	0·82	0·21	0·47

The amount of minerals required by the child after weaning is not known.

Water.—Owing to the greater proportion of water in the infant's body tissues (70 per cent. as against 60 per cent. in the adult), as well as to its more rapid metabolism, the infant requires a greater amount of water per unit of body-weight than does the adult. Thriving breast-fed babies are found on the average to take about 160 grm. of milk per kilogramme body-weight in a day, containing about 150 grm. of water. Hence we infer that the normal daily water requirements of a baby are about 15 per cent. of its body-weight, or about 2½ oz. per pound. Up to 3 months the requirements are 3 oz. per pound. On the same basis an adult weighing 10 stone would require as much as about 22 pints of water a day! Actually an adult takes no more than about one-fifth as much. The water requirement of a baby is an important point to remember in connection with artificial feeding of infants, inasmuch as an infant given too little water will, in spite of abundance of the other food constituents, cease to thrive, like a plant which has not been watered.

It is usual to take 3 oz. to the pound weight as the water requirement of an infant under 3 months, 2½ oz. to the pound between 3 and 6 months, and 2 oz. to the pound between 6 and 12 months, so that a 10 lb. baby who receives only 25 oz. of milk mixture must get an extra 5 oz. of water between the feeds.

The water is excreted as follows (the percentages are of the intake):—

By the kidneys ..	59 per cent.	(i.e., 88·5 grm. per kilogramme)
By the lungs	33 ,,	,, 49·5 ,, ,,
By the fæces	6 ,,	,, 9 ,, ,,

The remaining 2 per cent. (or 3 grm. per kilogramme) are retained.

Exact metabolism experiments have shown that in an infant of 5 kg., gaining 25 grm. per day, the gain is made up as follows:—

Protein..	3·13 grm.
Fat	3 ,,
Water	18 ,, [1]

[1] W. M. Feldman, "Child Physiology," p. 536.

Water Retention.—We see therefore that the gain is largely a matter of water retention. The amount of water retained is determined by the following other food elements: (1) The salts—which act by osmosis—the greater the amount of sodium chloride ingested, the greater the amount of water retained; this water retention, however, is only temporary, and disappears when the salt is withheld; (2) the carbohydrates, especially starch, also favour water retention, which is of a more lasting character. *Hence, infants fed on starchy food may show great gain in weight on account of excessive water retention.*

Some people recommend giving a little water in a feeding-bottle a couple of times a day, even to a breast-fed baby, in order to accustom it to bottle-feeding, so that it should take to the bottle readily when the time for weaning comes. Refusal to take a bottle when weaning begins is fairly common, and occasionally exceedingly troublesome, but such annoyance can be prevented by starting giving water in a bottle in early infancy. The 2 to 3 oz. of water per pound is the average quantity required by normal babies. Babies who lose water excessively by perspiration (e.g., in the hot summer, or in cases of pyrexia), or by the stomach and bowels (diarrhœa and vomiting), require correspondingly greater amounts.

Vitamins, or Accessory Food Substances.—Gowland Hopkins was the first to show, in 1912, that in addition to protein (containing the requisite amino-acids), fat, carbohydrate, water and salts, given to an animal in the requisite proportions as well as in amounts sufficient to satisfy the caloric requirements, there is still something else, the nature of which is not known, required to be included in the food, not only to make the animal grow but even to maintain its weight. Thus, if young rats are fed on a mixture containing *pure* caseinogen, fat, carbohydrate, salts and water, in proportions equivalent to those contained in milk and in adequate amounts, the animals cease to grow. If, however, a very minute quantity of some natural food substance, such as 3 c.c. of fresh milk a day, be added to the diet, the growth curve at once begins to rise and continues to do so, although the quantity of milk added does not increase by any appreciable amount the energy value or the body-building character of the food. Hence it is clear that fresh milk contains some substance or substances which stimulate growth, and that these substances are removed when the food is purified. These substances are called vitamins, or accessory substances.

Recent work has shown that certain diseases or trains of symptoms are due to the lack of certain vitamins, of which four are known—not because they have been isolated, but from the results of their absence in the diet. From the point of view of Child Hygiene, only three, viz., A, C and D (or A ii) vitamins and their corresponding three deficiency diseases are of interest to us here.

Vitamin A.—This substance, originally described by McCollum and his co-workers in 1913, is found in butter and yolk of egg, as well as in a large number of other foodstuffs, including the tissues of green chlorophyll-containing plants (i.e., fresh vegetables). It is specially abundant in cod-liver oil. Other animal fats contain it in smaller amounts and vegetable fats contain none of it. On account of its association with fatty substances, it has been named fat-soluble vitamin A. The researches of Mellanby have shown that deficiency of this, or a very similar vitamin, is most probably responsible for rickets, and it may therefore be called the antirachitic vitamin, although it is at present disputed that the antirachitic factor in cod-liver oil is really a vitamin, since irradiation of various foods, especially those containing cholesterol, with the mercury vapour lamp renders them antirachitic.

Deprivation of this vitamin also causes growth to stop, is responsible for a disease of the eye called xerophthalmia or keratomalacia, and interferes with the normal development of the teeth, causing delayed dentition and caries.

Recent work has shown that vitamin A really consists of two separate vitamins, which have been named A and D, or A i and A ii. **Vitamin A (or A i)** promotes growth and prevents xerophthalmia, whilst **vitamin D (or A ii)** is the antirachitic factor.

Vitamin C.—This substance is soluble in water, and is present in all fresh fruits, especially oranges, grapes, lemons, and other fresh vegetables such as cabbage, lettuce, green vegetables, potatoes, tomatoes, and also to a small extent in fresh milk. Its absence in the food is responsible for scurvy, and whilst ordinary cooking does not affect the antirachitic factor, such treatment destroys this antiscorbutic vitamin, and hence feeding on cooked foods alone causes scurvy.

Skimmed milk and condensed milks or milk powders contain no vitamin A.

Infants fed on cooked milk must have vitamin C supplied to them in the form of orange juice, or the juice of swedes or tomatoes.

Vitamin B is the antineuritic accessory factor, absence of which is responsible for beri-beri; and **vitamin D**, which is the antirachitic factor, has been mentioned under vitamin A.

Vitamin Requirement.—It is impossible in the present state of our knowledge, when the vitamins have not been isolated, to state in quantitative terms what are the vitamin requirements of any individual. It is possible, however, to state what is the relative richness of any particular food substance in any particular vitamin. Thus, vitamins A and D are most abundant in

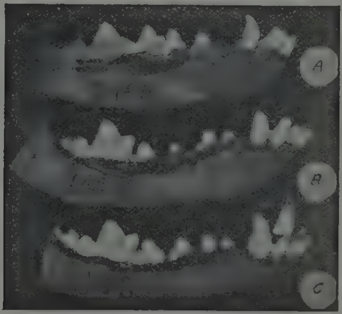

FIG. 86.—The effect of the calcifying vitamin. The jaws of three puppies of the same litter brought up on the same diet, except that A contained 10 c.c. of linseed oil daily, B contained 10 grm. of butter daily, and C contained 10 c.c. of cod-liver oil daily. Note the perfect formation of the teeth of C and the imperfectly formed teeth in A. (May Mellanby, *Brit. Med. Journ.*, March 20, 1926.)

butter, cod-liver oil and yolk of eggs, are present in appreciable quantities in cream, whole milk, carrots, nuts, &c., and are absent from vegetable oils (e.g., olive and cotton-seed oil), potatoes, oranges, &c.

Isolation of Vitamin D.—In 1926, Rosenhain and Webster, of the National Institute for Medical Research, isolated a substance from irradiated cholesterol which they call ergosterol, and which they believe to be pure vitamin D. One thousandth of a milligram of this per day given to a rat is sufficient to keep it in health, and the amount that a human being would require from birth to death would be approximately half an ounce.[1] If this discovery is confirmed, the vitamin so isolated will act as a very much more efficient and certainly much cheaper substitute for cod-liver oil and ultra-violet rays in the prevention and treatment of rickets.

[1] *Bioch. Journ.*, xx, 1926.

Infants and growing children require a relatively greater amount of vitamins than adults. Also the diet of pregnant and lactating women must contain a good supply of these accessory food factors, since the vitamins in the mother's blood or milk are not synthesised in her own body, as is the case with the other food constituents, but are derived unaltered from her food.

Edward Mellanby[1] fed two sets of pregnant and lactating bitches, that had been sired by the same male, on diets which were identical as regards the proportions of the essential elements as well as the caloric value, but differing with regard to the vitamin content. Thus, the diet of Group A contained cod-liver oil, whilst that of B contained the same quantity of olive oil. Both groups lived throughout the experimental period under identical conditions. After weaning, the puppies from each litter were given identical diets (deficient in antirachitic vitamin), and it was found that those of litter B presented more marked rickety changes in their bones than those coming from litter A.

The Influence of Vitamins on Teeth.—May Mellanby has shown that the structure of the teeth in growing puppies can be controlled experimentally by diet. A diet rich in the fat soluble vitamin will produce healthy and well-calcified teeth, whilst one deficient in that vitamin will produce badly-formed teeth (see fig. 86).

Good and Bad Diets.

R. McCarrison,[2] basing himself on the facts that certain Indian races, like Sikhs and certain Himalayan tribes, who are of exceptionally strong physique and live on diets consisting of coarsely ground whole-meal bread, milk, milk-products, tubers, roots, green leafy vegetables and fruit, with meat occasionally, has performed experiments on rats with the object of ascertaining whether there is any causal relationship between such a diet and physical development. Choosing two colonies of half-grown rats—each containing twenty animals of approximately the same age and of the same aggregate weight, he kept them under identical environmental conditions, with the exception that one colony received a diet corresponding to that eaten by Sikhs (hereafter called "good diet"), while the second colony were fed on a diet resembling that eaten by many Western people of the poorer classes, viz., white bread, vegetables cooked in water, coconut oil, tinned meat, tinned jam, tea and sugar (hereafter called "bad diet"). The coconut oil contained 100 gr. of boric acid to the pound—an antiseptic usually present in butter, margarine, cream, &c., used in western countries, and the tinned meat was exposed to formaldehyde vapour for some hours in order to imitate the Langley process of treating chilled meat for export.

[1] *Brit. Med. Journ.*, March 20, 1926.
[2] *Brit. Med. Journ.*, October 23, 1926.

After an experimental period of six months, McCarrison found the following results (see also fig. 87 (*a*) and (*b*)) :—

TABLE CXVIII.

	Colony fed on good diet	Colony fed on bad diet
Mortality	15 per cent. (3 out of 20)	45 per cent. (9 out of 20)
Causes of death ..	(1) Injury of abdomen (2) Pneumonia (3) Unknown	3 were killed and eaten by their fellows (showing that they were in a weakly condition) 6 died of broncho-pneumonia
State of gastro-intestinal tract	Healthy	Unhealthy
Survivors	Well grown, strong and active. Aggregate weight steadily rose	Ill grown, weakly and listless. Aggregate weight steadily fell (apart from losses by death)

From these experiments McCarrison concludes that a diet composed of whole-wheat, milk, milk-products, sprouted legumes, uncooked vegetables and fruit, with fresh meat occasionally, far surpasses in nutritive value that composed of white bread, tea, sugar, margarine, jam, boiled vegetables and tinned meat, to which the common food preservatives—boric acid and formaldehyde—are added. The bad diet leads to stunting of growth and to disease of the gastro-intestinal tract, as well as of the lungs.

The Bacteriology of the Gastro-Intestinal Tract of the Infant.—At birth the intestinal contents are sterile, but they soon become infected through the mouth and anus with fermentative and putrefactive organisms, and the character of the flora resulting from this invasion is afterwards determined by the nature of the food that the infant is given.

(*a*) *Breast-fed Infants.*—Since breast-milk, as we have seen, contains a high percentage of sugar (7 per cent.), as compared with that of either protein (1·5 per cent.) and fat (3·5 per cent.), the organisms which thrive in the intestines of breast-fed babies are those which grow best in carbohydrates and thrive in an acid medium. These are mainly of the Gram-positive variety, e,g., the *Bacillus bifidus* of Tissier. This bacillus finds a favourable environment in the breast-fed baby's intestine, since the lactic acid ($C_3H_6O_3$) produced from the fermentation of the lactose ($C_{12}H_{22}O_{11}$), as the result of the action of the fermentative organisms favours its growth, and inhibits the growth of the *Bacillus coli*, the *Bacillus lactis aerogenes* and other putrefactive, Gram-negative organisms. Hence, on microscopic examination

of the fæces of breast-fed babies, the *Bacillus bifidus* (showing antler-like ramifications) dominates the scene, and the reaction of such a stool is generally acid.

(*b*) *Artificially-fed Infants.*—In cow's milk the percentage of protein in proportion to carbohydrate is very high (70 per cent. to 80 per cent.) as compared with that in human milk (about 20 per cent.), and hence the Gram-negative *Bacillus coli*, as well

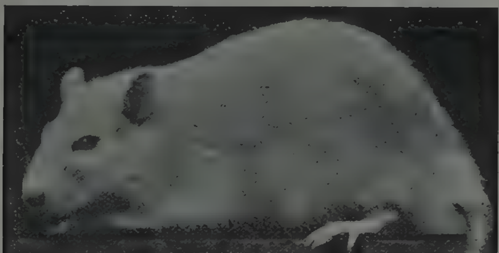

FIG. 87.—(*a*) Medium-sized rat from the colony fed for six months on the "good diet."

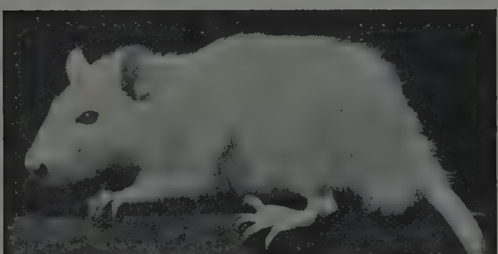

FIG. 87.—(*b*) Medium-sized rat from the colony fed for six months on the "bad diet." Same scale as fig. 87 (*a*) (from McCarrison, in *British Medical Journal*).

as proteolytic organisms, which thrive in an alkaline medium, make their appearance in the intestine. The peculiarity of the intestinal flora of an artificially-fed infant is the fact that there is no predominance of any particular organism. The reaction of such a stool is usually alkaline.

Escherich was the first to point out, in 1887, the direct *antagonism between the fermentative and putrefactive bacteria*, and he

also showed that it is possible to hinder putrefaction in a baby's intestine by increasing fermentation within it by giving the baby an excess of carbohydrate.

The Diet of Older Children.—So long as the infant's sole diet consists of milk, whether human or modified cow's milk, the problem of its nutrition is one of comparative simplicity, because such food already contains all the necessary ingredients in the proportions required by the child, and in forms suitable for the baby's digestive capacity. When, however, at about the end of the first year, the time arrives for the milk to be supplemented in a gradually increasing degree by some other article or articles of diet, the task of preparing a diet table which will fulfil all the necessary requirements becomes a problem of very considerable difficulty. All authorities are practically in agreement regarding the total approximate number of calories per unit of body-weight per day required by the average child at different ages, but there is some lack of unanimity with respect to the proper distribution of the calories between protein, carbohydrate and fat.

The Requisites of a Child's Diet.—A child's diet must answer the following requirements:—

(*a*) Those enumerated on pp. 334 and 335, regarding the requisites of an infant's diet:—

(1) It must be of suitable calorific value.

(2) It must contain protein, carbohydrate, fat, salts and water, in the requisite proportions.

(3) The protein must contain the necessary amino-acids or building stones.

(4) It must contain a sufficiency of vitamins.

(*b*) In addition:—

(5) It must be sufficiently solid to give the jaws, the masticatory muscles and the teeth the requisite amount of exercise, since insufficient work done by these structures during the period of growth and eruption of teeth results in imperfect growth of the jaws leading to the eruption of the teeth in an irregular and overcrowding manner, favouring dental decay. Moreover, as the jaws form the boundaries of the nasal cavities, defective growth of these bones results in the production of narrow nasal cavities, which in turn causes the child to become a mouth-breather and may also lead to the growth of adenoids.

(6) It must be of suitable digestibility not to overtax the child's somewhat delicate alimentary tract, and also to give

sufficient exercise of the gastro-intestinal tract to develop its secretory and muscular mechanisms.

(7) It must contain sufficient ballast to fill the bowels to enable them to contract and to drive on their contents. Lack of sufficient ballast may sow the seed for constipation in later life, because of the lack of training on the part of the bowels.

TABLE CXIX.—CALORIES PER KILO IN BOYS.

Weight in kilos (boys)	Calories per kilo								Total	
	Basal		Growth		Activity		Excreta			
	Absolute	Per cent.	Absolute	Per cent.	Absolute	Per cent.	Absolute	Per cent.	Per kilo	Per child
3	46	38	56	47	8	7	10	8	120	360
4	50	42	52	43	8	7	10	8	120	480
5	54	46	46	39	8	7	10	8	118	590
10	54	55	22	22	13	13	10	10	99	990
14	50	56	13	15	17	19	9	10	89	1,240
17	47	56	9	11	19	23	8	10	83	1,410
20	44	53	7	9	21	26	8	10	80	1,600
22	42	53	7	9	23	28	8	10	80	1,760
24	41	51	8	10	23	29	8	10	80	1,920
30	36	45	7	9	29	36	8	10	80	2,400
36	33	41	8	10	31	39	8	10	80	2,880
42	31	39	10	12	31	39	8	10	80	3,360
51	29	38	11	13	31	39	8	10	79	4,030
60	27	43	2	3	27	44	7	10	62	3,720
Adult	25	52	0	0	18	38	5	10	48	3,265

TABLE CXX.—CALORIES PER KILO IN GIRLS.

Weight in kilos (girls)	Calories per kilo								Total	
	Basal		Growth		Activity		Excreta			
	Absolute	Per cent.	Absolute	Per cent.	Absolute	Per cent.	Absolute	Per cent.	Per kilo	Per child
3	51	43	51	43	8	6	10	8	120	360
4	53	45	49	41	8	6	10	8	120	480
5	55	47	45	38	8	7	10	8	118	590
10	55	56	21	21	13	13	10	10	99	990
14	47	54	14	16	17	20	9	10	87	1,220
17	43	54	10	12	19	24	8	10	80	1,360
20	40	53	7	9	21	28	8	10	76	1,520
22	38	50	8	11	22	29	8	10	76	1,670
24	37	49	8	11	23	30	8	10	76	1,820
30	36	46	9	11	26	33	8	10	79	2,370
36	35	44	10	12	27	34	8	10	80	2,880
42	34	41	9	11	27	35	8	10	78	3,275
51	32	51	3	5	21	34	6	10	62	3,160
60 (adult)	25	57	0	0	15	33	4	10	44	2,640

(1) **Calories.**—We have already seen that the total daily need of calories by the growing child is made up of the four components : basal metabolism, muscular activity, growth quotient and fæcal loss. The preceding tables (after Holt and Fales)[1] give the absolute and percentage number of calories required under each of the four headings.

The basal requirement is based upon the results of the investigations of Benedict and Talbot. The growth quotient has been calculated from the normal average rate of growth. Rubner estimated that about 80 calories a day are needed to increase the weight of the human body by one kilogramme in one year. Hence, knowing the average gain in weight during any year (as given in anthropometrical tables stating the average weight at any year of age), the growth quotient during that year is easily calculated.

Dubois has given the following equation to express the basal metabolism (in calories per square metre of surface per hour) at various ages :—

$$\text{Log } y = 1\cdot8362 - 0\cdot0118\,x,$$
$$\text{where } y = \text{basal metabolism.}$$
$$x = \text{age.}$$

The following table (Table CXXI) gives the observed and calculated results in a series of twenty-seven boys.

TABLE CXXI.

Mean age of boys in years (x)	Basal metabolism in calories per square metre per hour (y)	
	Actual	Calculated
6·5	57·5	57·6
12·6	50·4	48·7
13·7	49·4	47·3
16·5	43·0	43·9
19·25	40 7	40·7

According to Dreyer[2] the basal metabolism may be expressed by the formula $C = \frac{W^{0\cdot5}}{K \cdot A^{0\cdot1333}}$,

where C is the number of calories produced in four hours
A ,, age in years
W ,, weight
and K is a constant whose value is 0·1015 for boys and 0·1127 for girls.

The allowance for activity necessarily varies with the age and temperament of the child. A very active child will require much more than a quiet child. The actual number of calories

[1] L. Emmett Holt and Helen L. Fales, *Am. J. Dis. Ch.*, 21, 1921, p. 16.
[2] *Lancet*, ii, 1920.

to be allowed for activity during any age interval can only be hypothetical. Between the second and thirteenth year the activity quotient steadily increases from 7 per cent. to 44 per cent.; after that, when growth becomes more active, it becomes stationary and afterwards decreases.

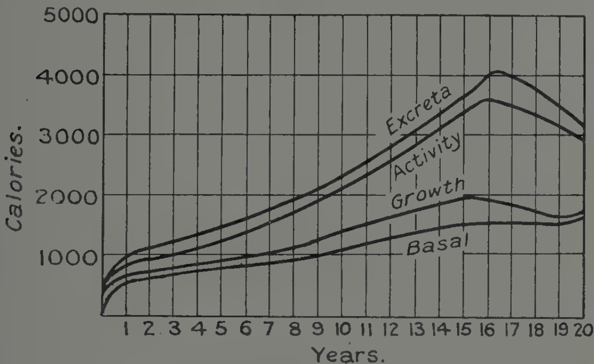

FIG. 88.—Total calories for boys (after Holt).

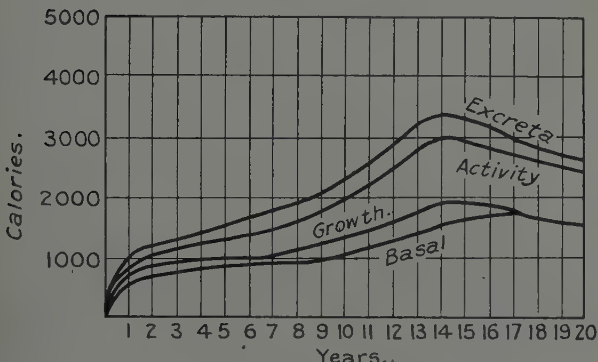

FIG. 89.—Total calories for girls (after Holt).

From the tables (Tables CXIX-CXXIII, and charts,[1] figs. 88-92) we see that the average caloric requirement is about 100 calories

[1] In using these charts it must be remembered that children below the normal weight require proportionately more food than those of normal weight, and vice versa.

The spaces between the various lines in each chart, from the base line upwards, represent the allowance for the different factors which make up the total.

per kilogramme at 1 year. It then falls in the case of boys to about 80 at 6 years, and remains stationary at this value up to the age of 15. It then steadily falls until it reaches the adult level of about 48. In the case of girls the requirement falls from 100 calories per kilogramme at 1 year to 74 at 9 years. It then rises slightly till the age of 13 (80 calories a day) and then gradually declines and reaches the adult level of 44 calories a day per kilogramme.

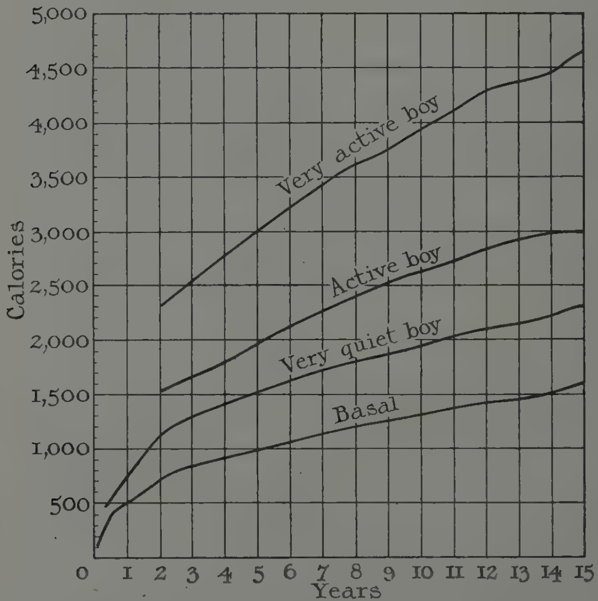

FIG. 90.—Influence of muscular activity on metabolism.

In round numbers one may say that boys during school age require *80 or more* calories per kilogramme body weight per day and girls during the same age period need *up to 80* calories per kilogramme.

It is to be remembered that the caloric requirement must be computed from the weight rather than from the age, so that children who are under weight for their age require more and

those who are over weight require less than those who are of normal weight.

It must further be noted that these figures represent normal averages and may have to be modified in individual children. The most variable factor is that of muscular activity, which must be taken into account in individual cases (see fig. 90). The tables are useful, however, in catering for large numbers of children in institutions.

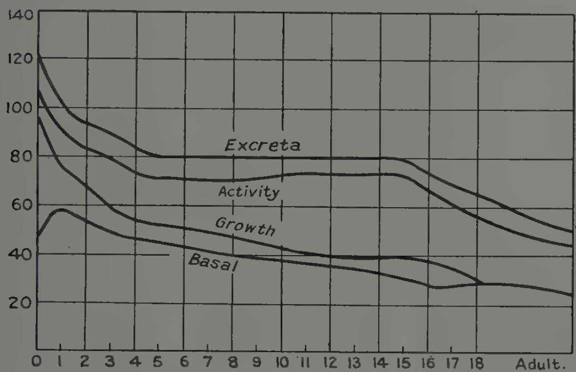

FIG. 91.—Calories per kilo for boys (after Holt).

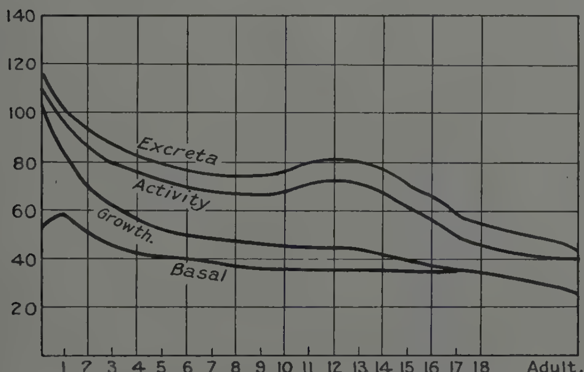

FIG. 92.—Calories per kilo for girls (after Holt).

The following tables (Tables CXXII and CXXIII) give the number of calories under the various headings for different ages in boys and girls :—

TABLE CXXII.—BOYS.

Age	Average weight in lb.	Calories per lb. for Basal metabolism	Growth	Activity	Loss in excreta	Total	Total calories per day for child of average weight
3-4	34·34	23	3·0	7·7	4·1	37·8	1,298
4-5	37·22	22	2·8	8·2	4·1	37·1	1,381
5-6	40·46	21	3·2	9·1	3·6	36·9	1,493
6-7	44·32	20	3·4	9·5	3·6	36·5	1,618
7-8	49·20	19	4·6	10·5	3·6	37·2	1,830
8-9	53·95	19	2·6	10·5	3·6	35·7	1,926
9-10	58·12	18	2·7	11·4	3·6	35·7	2,075
10-11	63·38	17	3·4	12·7	3·6	36·7	2,326
11-12	69·53	16	3·3	13·2	3·6	36·1	2,510
12-13	75·03	15	2·3	13·6	3·6	34·5	2,588
13-14	80·70	15	3·0	14·1	3·6	35·7	2,881
14-15	89·07	14	4·1	14·1	3·6	35·8	3,189

TABLE CXXIII.—GIRLS.

Age	Average weight in lb.	Calories per lb. for Basal metabolism	Growth	Activity	Loss in excreta	Total	Total calories per day for child of average weight
3-4	33·33	21	3·6	7·7	4·1	36·4	1,213
4-5	36·36	20	2·8	8·6	3·6	35·2	1,273
5-6	39·49	19	3·2	8·6	3·6	34·4	1,358
6-7	43·08	18	3·1	9·5	3·6	34 2	1,473
7-8	47·24	17	3·5	10·0	3·6	34·1	1,611
8-9	51·82	17	3·2	10·4	3·6	34·2	1,762
9-10	56·39	16	2·9	10·9	3·6	33·4	1,883
10-11	62·10	16	4·0	11·4	3·6	35·0	2,173
11-12	69·69	16	4·3	11·8	3·6	35·7	2,478
12-13	77·11	16	3·3	12·3	3·6	35·2	2,714
13-14	84·24	16	3·3	12·3	3·6	35·2	2,965
14-15	92·17	15	3·2	11·8	3·2	33·2	3,060

These tables, as just pointed out, must not be used slavishly. They merely represent averages, and each child must be supplied with the number of calories required to satisfy its own special needs with regard to its environment, the season of the year, the climate in which it lives, its temperament (placid or nervous), activity and its weight for its age. An underweight child requires proportionately more and an overweight child requires proportionately less than does a child of average weight for its age.

Relative Proportion of Protein, Fat and Carbohydrates.

Protein.—Whilst carbohydrates and fats are, as we have seen (p. 345), to a certain extent interchangeable, protein cannot be replaced, since in virtue of its nitrogen content it is indispensable not only for the constant renewal of protoplasmic waste, but, in the case of children, it is also essential for the building of new tissue. It must be remembered, however, that not all the proteins yield on digestion all the various amino-acids which are required for growth. Hence special care must be taken to provide the child with the proteins which contain large proportions of the necessary amino-acids, or if proteins are given which contain smaller proportions of these substances, they must be supplied in the diet in greater amounts. It is clear, therefore, that the amount of protein required by the growing child is, to a great extent, determined by the kind of protein given. As animal proteins resemble human proteins more nearly than do those of vegetable origin, they are undoubtedly preferable during the growing period.

The exact amount of protein required by a child is not definitely settled. Schlossmann and Sommerfeld apply to the child Chittenden's teaching regarding a lower protein requirement by the adult, and advocate a smaller protein intake than is generally found to be the case from analyses of large numbers of children's dietaries. They believe that 2 grm. of protein per kilogramme is sufficient for any normal child, and that this amount should be decreased as the child gets older.

The average minimum protein intake in the case of adults of average weight has been found, by Sherman, to be at the rate of 0·6 grm. per kilogramme, and he concludes that for maintenance an intake of 1 grm. of protein per kilogramme is ample in the case of an average adult. As, however, a child's metabolism is considerably more active, Holt believes that its protein requirement for maintenance should be about 1·5 grm. per kilogramme, and although certain vegetable proteins such as zein (from corn) are deficient in the amino-acid lysin which is necessary for maintenance, yet on the whole it is possible to supply the necessary maintenance protein from the vegetable world. It must be remembered, however, that whilst as much as 97 per cent. of animal protein is digested and assimilated, no more than about 60 to 80 per cent. of vegetable protein is assimilable. Indeed, the more nearly the composition of a food approaches that of our own tissue the more readily is it digested

and absorbed. The protein required for growth cannot easily be supplied from the vegetable world. It is true that the breast infant thrives well on 1·5 grm. of protein per kilogramme, but it must be remembered that the caseinogen and lactalbumin of human milk together contain in abundance all the amino-acids required both for maintenance and growth. When, however, the child is put on a mixed diet and deprived of human milk, the total amount of protein per kilogramme has, as is found in the case of infants fed on cow's milk, to be approximately doubled. We may therefore accept 3 to 4 grm. of protein per kilogramme in the case of younger children and 2 grm. per kilogramme in the case of older ones. The exact amount must vary directly with the activity of the child.

TABLE CXXIV.

Age	Boys					Girls				
	Protein		Fat	Carbo-hydrate	Total	Protein		Fat	Carbo-hydrate	Total
	(A.)	(V.)				(A.)	(V.)			
3-4	139	65	453	650	1,307	121	62	423	607	1,213
4-5	138	69	484	690	1,381	127	64	445	637	1,273
5-6	149	75	523	746	1,493	136	68	475	679	1,358
6-7	162	81	566	809	1,618	147	74	516	736	1,473
7-8	183	92	640	915	1,830	161	81	563	806	1,611
8-9	192	96	675	963	1,926	176	88	617	881	1,762
9-10	207	104	727	1,037	2,075	188	94	659	942	1,883
10-11	233	117	813	1,163	2,326	217	109	760	1,087	2,173
11-12	251	126	878	1,255	2,510	249	130	865	1,244	2,488
12-13	259	130	905	1,294	2,588	271	136	950	1,357	2,714
13-14	288	144	1,009	1,440	2,881	296	148	1,038	1,483	2,965
14-15	319	160	1,111	1,599	3,189	306	153	1,071	1,530	3,060

(A = animal. V = vegetable).

We arrive, therefore, at the following **conclusions** :—

(1) Total protein requirement of young children = 4 grm. per kilogramme.

(2) Maintenance protein = 1·5 grm. per kilogramme.

(3) Growth protein = 2·5 grm. per kilogramme.

(4) The maintenance protein can be supplied from the vegetable world, but that required for growth must come from animals. In other words, the proportion of animal to vegetable protein must be about 2·5 : 1·5, or about 60 per cent. of the total protein must come from the animal world. It will further be

clear that if for economical or other reasons it is necessary to give less than 60 per cent. animal protein, a much larger proportion of vegetable protein would be required to supply the amino-acids necessary for growth.

That lack of proteins gives rise to defective growth is made probable from the height and weight curves of Japanese children. During infancy all Japanese babies are breast-fed, and there is practically no difference in the heights and weights of Japanese babies as compared with those of European or American infants. After infancy, however, when Japanese children receive very little milk, meat, or cheese, but are fed mostly on rice, vegetable oils, fish, sweets and vegetables, and therefore receive a deficiency of protein, the growth in height and weight becomes very slow. That this stunting of growth is not entirely an hereditary character is shown by the fact that Japanese children in the United States are taller and heavier than those of corresponding ages living in Japan. On the other hand, great excess of proteins, especially in ailing children, may result in: (1) deprivation of minerals, because the acids formed as the result of protein digestion combine with the alkaline carbonates in the blood and are excreted as such; (2) surcharge of the blood with purin bodies, giving rise to uric acid, which, although normally excreted in its entirety in the urine, may, according to French authorities, accumulate in the body and give rise to various manifestations analogous to gout in adults; (3) excessive intestinal putrefaction with the production of toxic bodies. The danger of this, however, is minimised in the presence of sufficient carbohydrate.

Fat.—The metabolism and function of fat in the diet has been discussed on p. 342. As regards the amount of fat required by the young child after the age of infancy there are no exact scientific observations. Analysis of the diets of large numbers of children, however, shows that the large majority take between 2 and 4 grm. per kilogramme daily, with an average of about 3 grm. per kilogramme. As the child gets older the requisite fat ration diminishes. On the other hand, some authorities believe that fat is not an essential article of diet and that it can be replaced by carbohydrate without harm (Pirquet). It is, however, generally admitted that although no immediate bad consequences result from such substitution, so that Pirquet's system is not harmful as a temporary measure, continuous lack of fat in the diet increases the child's susceptibility to tuberculosis and, in the case of infants, to rickets and other

diseases. This greater susceptibility to disease, on the part of children brought up on excess of carbohydrate, is due to the greater water retention in their bodies, since the experiments of Weigert have shown that tissues containing large proportions of water form good culture media for pathogenic bacteria.

In conclusion, one may say that the proper daily amount of fat to be allowed to young children is 4 grm. per kilogramme (= 0·4 per cent. of body-weight) at 1 year, and about 3 grm. per kilogramme (= 0·3 per cent. of body-weight) at 6 years and thereafter.

Carbohydrate.—The amount of carbohydrate to be taken must be such as to make up the requisite daily number of calories. We have seen that a child between the second and sixth year requires 4 grm. of protein and 4 grm. of fat per kilogramme body weight per day. These amounts yield $4 \times 4 + 4 \times 9 = 52$ calories per day. But such a child requires 100 calories per kilogramme a day. Therefore 48 calories must come from carbohydrate. This means that the daily intake must amount to 12 grm. per kilogramme. At 6 years and thereafter, the amount works out at 10 grm. per kilogramme. Hence we see that between the second and sixth years of life the child's diet should contain:—

4 grm. of protein		per kilogramme per day.
4 ,, of fat		
12 ,, of carbohydrate		

The average distribution of calories will then be (in round numbers):—

Protein	15 per cent.
Fat	35 ,,
Carbohydrate	50 ,,

In the case of adults the usual daily ration is 100 grm. of protein, 100 grm. of fat, and 400 grm. of carbohydrate per person (i.e., 1·4 grm. protein, 1·4 grm. fat and 5·6 carbohydrate per kilogramme body weight), with the following percentage distribution of calories: Protein, 15 per cent.; fat, 30 per cent.; carbohydrate, 55 per cent.

At 6 years and thereafter, the requirements are:—

Protein, 3 grm.	per kilogramme body-weight.
Fat, 3 grm.	
Carbohydrate, 10 grm.	

The average distribution of calories is of course approximately the same as during the age period 1 to 6, viz.: Protein, 15 per cent.; fat, 35 per cent.; carbohydrate, 50 per cent.

If the child does an unusual amount of exercise, the amount of carbohydrate must be increased.

Kind of Carbohydrate.—Carbohydrates are of two kinds: *Soluble* (e.g., cane sugar, lactose, fructose, glucose, maltose, &c.), and *insoluble* (starch, as found in potato, bread, oatmeal, rice, &c.). Even the youngest baby has the enzymes for converting starch into sugar, but until the baby is a few months old these enzymes are present in small quantities. After the age of 6 months it is quite safe to give the baby some starch, and after the age of 1 year it is found that the two kinds of carbohydrates may be given in about equal proportions.

Formation of Sugar out of Protein and Fat.—Sugar is formed in the body, not only out of carbohydrates but also out of protein and fat. If a dog fed entirely on protein is given an injection of phloridzin it will begin to pass sugar in the urine. This sugar comes from the ingested protein, and it has been estimated that each gramme of protein is capable of producing 0·58 grm. (i.e., 58 per cent.) of sugar.

The researches of Lusk and others have shown, however, that it is not all the amino-acids which are capable of producing sugar. Thus, whilst glycocoll, alanine and cystine can produce sugar, valine, leucine, lysine, histidine, tryptophane, &c., are incapable of doing so.

Sugar can also be produced out of the glycerine moiety of fats. As, however, glycerine does not represent more than about one-tenth of the weight of the fat molecule, and as most of the glycerine reunites with the fatty acid with the re-formation of fat, the amount of carbohydrate coming from that source is negligible.

Relation between Fat and Carbohydrate.—Fat is said to burn in the body in a flame of carbohydrate, and in the absence of sufficient carbohydrate the combustion of fat is incomplete, resulting in the production of ketone bodies (ketosis or acidosis). It is believed that 1 grm. of carbohydrate is required for the complete combustion of 3 grm. of fat. We have also just seen that the metabolism of protein results in the formation of about 60 per cent. of carbohydrate. Hence a child taking, say, 4 grm. of protein per kilogramme body-weight will produce $\frac{4 \times 60}{100} = 2{\cdot}4$ grm. of carbohydrate. This would be capable of burning up $2{\cdot}4 \times 3 = 7{\cdot}2$ grm. of fat. Now the 4 grm. of protein produce $4 \times 4 = 16$ calories, the 7·2 grm. of fat produce $7{\cdot}2 \times 9 = 64{\cdot}8$ calories. Hence the protein and fat together will furnish the body with $16 + 64{\cdot}8 = 80{\cdot}8$, say 81 calories. But the child requires 100 calories per kilogramme, therefore we must produce the other 19 calories either from carbohydrate alone (5 grm.) or from carbohydrate and fat in the proportion of 1 : 3 by weight (since 1 grm. of carbohydrate is required to combust 3 grm. of fat). If we call the amount of carbohydrate x, then the amount of fat will be 3x.

Now x grm. of carbohydrate will yield 4x calories
3x ,, fat ,, 27x ,,
Therefore we have—
$4x + 27x = 19$ calories
i.e., $31x = 19$, so that $x = 0{\cdot}6$.

Hence the 19 calories can be made up out of 0·6 grm. of carbohydrate and $0·6 \times 3 = 1·8$ grm. of fat.

In other words, from the point of view of prevention of ketosis, it is theoretically possible to feed a child on 3 grm. of protein, $7·2 + 1·8 = 9$ grm. of fat, and 0·6 grm. of carbohydrate.

In practice, however, a child's intestines could not cope with so much fat, and experience shows that the proper distribution of the ingredients is that given above (p. 366), viz. : Protein, 4 grm. ; fat, 4 grm. ; carbohydrate, 12 grm. per kilogramme.

Water.—We have seen that the infant's body contains about 70 per cent. of water as compared with 60 per cent. in the adult's body. Children contain intermediate percentages which gradually diminish with the age of the child. The child takes its water both in the form of drink as well as part of the so-called solid foods which contain variable (10 per cent. to 90 per cent.) amounts of water. Thus, bread contains 40 per cent., raw meat 50 per cent. to 70 per cent., and raw fruit 70 per cent. to 90 per cent. of water. In addition, some water is formed in the body as the result of oxidation of the various foodstuffs. Thus, when fat is metabolised the following chemical reaction occurs :—

$$C_{57}H_{104}O_6 + 80O_2 = 57CO_2 + 52H_2O$$

In other words, 884 grm. of fat yield 936 grm. of water, or there is approximately 1 grm. of water produced from each gramme of complete metabolised fat. In the case of carbohydrate, the equation

$$C_6H_{12}O_6 + 6O_2 = 6CO_2 + 6H_2O$$

shows that each grm. of carbohydrate yields approximately 0·6 grm. of water.

Even protein yields water after it has been metabolised. The quantities of water so produced, however, are insignificant, for, as we have seen, protein and fat are taken in proportion of only about 0·2 per cent. of the body weight, whilst water is required to the extent of about 20 per cent., so that no more than about $\frac{1}{100}$ of the total water requirement can come from the metabolism of the organic food ingredients.

The total requirement of water varies with the amount of bodily work, the temperature of the surroundings and the nature of the food (e.g., salt content, &c.). In general the child's thirst forms the best guide for its daily requirements of water.

The water ingested is excreted from the body as follows : By kidneys (urine), 60 per cent. ; skin (sweat) and lungs, 33 per cent. ; fæces, 6 per cent. ; only 1 to 2 per cent. of the intake is retained in the body.

The nature of the food has an influence upon the amount of water retained in the body. Thus, as we have seen on p. 350, carbohydrates and salts will retain a lot of water in the body. Of the salts, sodium chloride retains the greatest amount of water. But whilst the water retention due to salts is transitory, that due to carbohydrates is of a more lasting nature.

Summary.—Most people are agreed that protein ought to be given in the proportion of 0·4 per cent. of the body-weight between the ages of 1 and 6 years, and of 0·3 per cent. of the body-weight after that age. There is not quite the same agreement with regard to the quantities of fat and carbohydrate, but the bulk of the evidence goes to show that the amount of fat should, during the growing period, be approximately the same as that of protein, and that at any age of childhood the distribution of the calories should be: Protein, 15 per cent.; fat, 35 per cent.; carbohydrate, 50 per cent. If this balance is departed from to any large extent various undesirable results may follow. Deficiency of protein results in stunted growth. Deficiency of fat, or of carbohydrate, may result in various forms of indigestion. Thus, low fat with high carbohydrate may cause not only diarrhœa and rickets but, on account of the water retention, predisposition to tuberculosis, &c. High fat with low carbohydrate tends to result in acidosis, because there is not enough carbohydrate to burn up the fat completely.

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CHAPTER XIV

BREAST-FEEDING

"Twas a Sage said it, and the Saying's good,
The Mother's Milk's the only Wholesome Food.
Large Meals upon the Sucking Babe bestow,
And, freely let the Snowy Fountains flow . . .
Life's fed with Life itself, and Blood with Blood.
. . . .
Have you not heard it in the Cradle cry,
And seen the ready Nurse to feed it, fly?
How soon it Laughs to see the Swelling Breast,
Seizes the Nipple, and returns to Rest?"
SCEVOLE DE ST. MARTHE, "La Pædotrophia" (1584).[1]

ONE of the very remarkable wonders of Nature is the way she prepares food for the infant. For the child's proper development it is very desirable that it should, during its early life, be constantly in the company of its mother, and Nature has therefore provided that the mother should in her own body have a food laboratory from which to nourish her infant. Still more wonderful is the fact that as the child grows and requires different food combinations in different quantities, so also does the amount, and to some extent the composition, of its mother's milk change at the same time. The milk of one woman is, therefore, not quite suitable for the baby of another, unless the baby of the one is of approximately the same age as that of the other.

With the history of breast-feeding I have dealt at sufficient length in the second chapter. It has from time immemorial been recognised that the mother's milk is the only one food which is physiologically suited for her infant. It forms the physiological connection between the mother and her infant, although the anatomical connection between them has been severed at birth. In Chapter V I have also given statistics demonstrating the advantages to the baby of breast- as compared with bottle-feeding. It has been stated, with some truth,

[1] Translated into English by H. W. Tylter, "The Art of Nursing and Rearing Children": A Poem in 3 Books. London, 1797.

that "the feeding-bottle has killed more infants than gunpowder has killed adults" (N. Guillot), but we may recapitulate some of the statistics here in a somewhat different way.

About 25 per cent. of humanity throughout the civilised world die during the first year of life. Of these deaths, 60 per cent. are due to digestive disturbances. In other words, 15 per cent. of all civilised people die during the first year of life of ailments connected with feeding. The mortality of the first year is about sixty times as high as during the fifteenth year, and is the same as at about eighty-five years. Moreover, apart from the immediate danger to the life of the child, improper feeding renders the surviving child liable to rickets, scurvy and general malnutrition, and their consequences. In this chapter I shall deal with some of the numerous matters in connection with the science and art of breast-feeding.

The Physiology of Lactation.—The breast consists of two distinct structures, viz., glandular cells or *parenchyma*, and supporting connective tissue or *stroma*. In addition, it contains blood-vessels, lymphatics and nerves, and the whole breast is, of course, covered with skin and subcutaneous fatty tissue. Milk is formed by the parenchymatous portion out of the ingredients in the blood brought to it by the blood-vessels. When the transformation is complete, the milk passes drop by drop from each cell into ducts which open into the nipple. For the performance of this work of transformation of blood into milk the glandular cells must, it is believed, be stimulated by some internal secretion or hormone possibly coming from the corpus luteum of the ovary, although the exact source of the hormone is not known with any certainty. The fact that the mammary gland enlarges but does not secrete during pregnancy, and starts secreting immediately after parturition, suggests that the fœtus or placenta may have some inhibitory effect upon the secretory process. The fact, however, that the mammary glands in marsupials develop before the placenta shows that the latter cannot be responsible for mammary secretion. Miss Lane-Claypon and Starling found that injection of fœtal extract into a virgin rabbit caused enlargement of the mammary glands, but when injected into lactating animals the secretion stopped. Similar results have been obtained with extracts of placenta (Mackenzie). On the other hand, injections of corpus luteum extract into virgin rabbits causes enlargement of the breasts together with secretion of milk. From these experiments it would seem, therefore, that both the fœtus as well as the

placenta contain two separate kinds of internal secretions, one which stimulates the growth of the gland, and another which inhibits the secretion of milk; and that the corpus luteum contains hormones which stimulate both the growth and the secretion of the gland. The fact that in egg-laying mammals the mammary glands develop after the laying of eggs, disposes of the theory alleging a causal relationship between a fœtal hormone and mammary secretion. As stated above, however, the exact source of the secretory hormone is not known, since cases are known where lactation has occurred not only in virgins but also in males, and the occurrence of lactation in new-born babies of either sex is quite a frequent phenomenon. This, however, can be explained on the supposition that the hormone came from the placenta (see p. 292).

The actual formation of milk is independent of the nervous system, since not only have the nerves leading to the mammary glands been divided without stopping the secretion, but stimulation of these nerves in non-lactating animals has failed to bring about secretion. On the other hand, once lactation has been started by means of various hormones, it is maintained by stimuli carried along by the sensory nerves coming from the nipples. Indeed, some even go as ar as to say that all breasts can at all times produce milk if sufficient stimulus is applied to the nipples, and the facts mentioned above regarding the occurrence of lactation in virgin girls or in males, to whose breasts were put sucking babies, afford evidence in support of this view. Anyway, there is no doubt whatever that a feebly lactating breast will be stimulated to greater secretion by the sucking of a strong infant, and on the other hand lack of sucking will cause the milk to disappear from a well-secreting breast. It is also a fact that some psychological stimulus has a great influence on mammary secretion. Thus, a placid mother eager to suckle her baby will generally have enough milk to satisfy her wish, whilst a nervous mother who is obsessed with the fear that she will be unable to nurse will have a very poor milk supply. Similarly, a sudden shock or other emotional disturbance, such as the hearing of bad news, fear of coming danger (a common cause in the time of air-raids during the war), &c., interferes with the process of lactation.

From what I have said it will be seen that the three requisites for a proper and adequate milk secretion are: (1) An adequate number of mammary gland cells; (2) a proper mam-

mary blood-supply containing the necessary hormones ; (3) proper nerve stimuli, (*a*) peripherally from the nipple, and (*b*) centrally from the brain. When any one or more of these factors is at fault the supply of milk becomes abnormal.

The breast, for instance, may contain very little parenchyma, although it may be rich in connective tissue and fat which make it appear very large. Such a breast will secrete very little milk. On the other hand, a small breast, deficient in stroma but rich in glandular tissue, will produce an adequate supply of milk. The size of the breast, therefore, gives no information regarding its secretory ability.

The blood-supply is rarely at fault quantitatively in connection with lactation, but it may be poor in that subtle hormone which stimulates the secretory cells. But, as we have stated, lack of proper nerve stimuli either from the nipple or from the brain will certainly interfere with the supply of milk.

Energy of Lactation.—The average number of calories taken by a nursing mother (70 kg.) per day is 3,000. The amount of breast milk taken daily by her infant (7 kg.) contains about 700 calories. In other words, the amount of energy spent by a mother in producing milk for her baby constitutes about 23 per cent. of the whole of the intake. Hence a nursing woman should take about one-fifth to one-quarter more calories a day than a non-lactating woman, and the distribution of the calories in the extra ration should be approximately that of human milk.

Galactagogues.—A galactagogue means a substance which when administered to the mother will help the process of lactation or milk formation. Numerous galactagogues have been put on the market, e.g., pituitary extract, corpus luteum, lactagol, &c., but there is no scientific evidence that they produce any appreciable results. Various kinds of food and of drugs have from times of antiquity onwards been recommended as galactagogues, and dugs and udders have been prescribed by Thomas Phaer (the father of English pædiatrics) in the sixteenth century, but the following remarks by John Rubräh[1] are both pithy and very pertinent. "The search for galactagogues," says Rubräh, "has been a quest of all ages ; the fountain of youth, the alchemists' gold, the golden fleece of Jason, have temporarily been withdrawn from the scene, but the quest of a good milk stimulant is still with us. The use of dugs and udders suggests our organic therapy. In a delightful play of yesterday, 'Peaceful Valley,' the inimitable Sol Smith used to tell how, in his childhood, he

[1] "Pediatrics of the Past," New York, 1925, p. 159.

cried for the moon. His mother, a wise woman, would pull down the blind and give him a big, round soda cracker and he was satisfied. The early physicians, and alas, those of to-day, often use the same method If they want a therapeutic agent and have it not, they use something and imagine they have it."

The good effects occasionally witnessed after the administration of such galactagogues as lactagol are most likely psychological rather than physiological. Pituitary extract undoubtedly produces a flow of milk from the breasts ; this, however, is not due to increased secretion but to the action of the pituitrin on the unstriped muscle-fibre of the breast causing them to contract and expel the milk already in the breast. Massage and manual pressure will do the same.

The only way to ensure a good flow of milk lies in :—

(1) Complete emptying of the breasts, by manual expression if necessary, after each feed. This was a method described by Soranus, of Ephesus.

(2) A healthy mode of life in the way of good nourishing food, neither too little nor too excessive to upset digestion, a plentiful supply of fluid, a reasonable amount of exercise, and above all placidity of mind.

The following is the routine adopted by Truby King for the re-establishment of breast-feeding in cases where ordinary means have failed.

5.45 a.m.	Mother wakened and given a glass of hot water, an orange, or half an apple.
6.0 ,,	*Suckle baby* in bed—after he has been weighed by nurse.
6.20 ,,	Complete emptying of breast by hand.
7.15 ,,	Rise, cold bath, or sponging all over followed by brisk rub down and special trunk exercises (see fig. 71, p. 272).
8.0 ,,	Short, active walk.
8.15 ,,	Breakfast.
8.45 ,,	Bath and dress baby.
10.0 ,,	Drink glass of water and *suckle baby*.
10.30 ,,	Bathe both breasts with hot and cold water alternately, and rub down with dry towel.
10.40 ,,	Massage of both breasts by nurse, or (after training) by mother.

11.0 a.m. Exercise (e.g., walking, strolling round the garden), relaxation (sewing, reading, writing, &c.) mostly in open air, in comfortable, warm, well-ventilated room in winter.

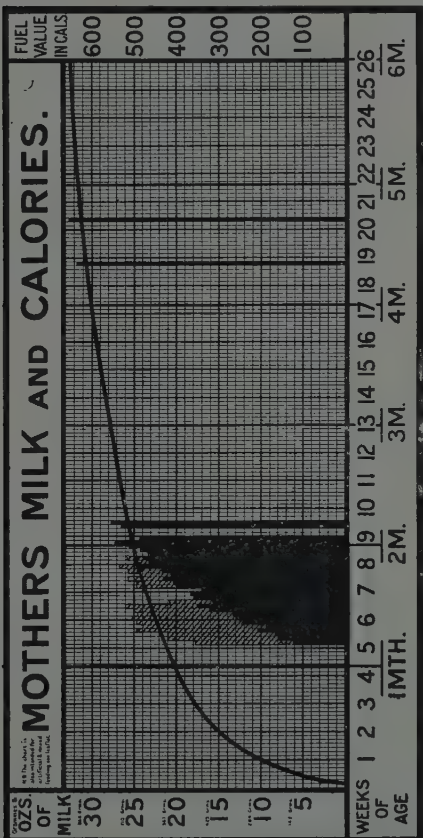

FIG. 93.—Restoration of breast-milk after weaning. The black columns indicate the amount of mother's milk; the cross-hatched columns represent the supplemental cow's milk. It will be seen that the supplementary milk required gradually diminished from about 12 oz. at the beginning of the fifth week to zero at the tenth week.

FIG. 93.

12.30 p.m. Dinner.
1.0 ,, Rest with feet up.
2.0 ,, *Suckle baby.*
3.0 ,, Cup of tea.

4.45-5.15 p.m.	Prepare baby for bed.
5.30 ,,	Supper.
6.0 ,,	*Suckle baby.*
6.30-8.30 ,,	Exercise and recreation in open air.
8.30 ,,	Bathe and massage breasts.
9.0 ,,	Prepare for bed.
9.30 ,,	Glass of hot milk, or milk and gruel.
10.0 ,,	*Suckle baby.*

The baby is weighed before and after each breast feed and the difference between the amount it should get and that taken from the breast is made up by means of modified cow's milk.

The accompanying chart (fig. 93) shows the result of such treatment (without any galactagogues). The mother's milk, in the case shown in the chart, increased from a little over 1 oz. on the first day to 21 oz. on the seventh day, and the supplementary modified cow's milk was diminished from about 20 oz. to *nil.*

The Chemistry of Human Milk.

From the physiological standpoint the mammary secretion must be considered at two different periods, viz.: (1) That occurring during the first few days of lactation and known as colostrum, and (2) that occurring subsequently until the end of lactation, i.e., milk proper. The transition from the one into the other takes place gradually. Both colostrum and milk contain all the various proximate principles, viz., protein, fat, carbohydrate, salts and water, rendering them complete foods. They also contain vitamins.

(1) *Colostrum.*—The colostrum milk is yellow, the colour being due to a pigment which adheres to the fat, and is more viscous than milk. It further differs from milk in having a high protein and low carbohydrate and fat contents. It also contains a number of large cells known as colostrum corpuscles. These are 10 to 20 μ in diameter ($\mu = \frac{1}{1000}$ of a millimetre) and their significance is not known with any certainty. The following table (Table CXXV) represents the relative compositions of colostrum and milk :—

TABLE CXXV.

	Protein	Fat	Sugar	Ash
Colostrum	9·8	2·6	2·9	0·40
Milk	1·5	3·5	7·0	0·20

The colostrum gradually changes in composition from day to day, until about the fifth day when it becomes practically indistinguishable from true milk. It also increases in amount from day to day.

Margaret F. Lowenfeld,[1] working in conjunction with Sybil T. Widdows, Muriel Bond and Effie T. Taylor, in an investigation of the factors which influence the chemical composition of human colostrum, found that:—

(1) There is a difference between the colostrum of primiparæ and multiparæ, especially with regard to protein.

(2) High initial values of protein, up to 8 per cent., are generally found in primiparæ.

(3) The protein falls steeply during the first week to reach the level normal for ordinary human milk (0·98 to 1·3 per cent.).

(4) The sugar content varies inversely with the protein. It is lowest during the first couple of days (4·4 per cent.) and steadily rises to reach the normal for milk (6·5 per cent.) by the end of the second week.

(5) The fat content is practically constant from day to day, but that it varies inversely with the quantity of fluid in the breast and directly with the degree of pressure exerted in extracting the milk.

(6) The ash content varies in the same way as the protein. It is as high as 1·2 per cent. in the early days and falls to 0·25 per cent. at the end of the first week.

(7) The protein content is somewhat higher at the end of a feed.

(8) The lactose content is somewhat higher at the beginning of a feed.

The Value of Colostrum to the New-born Infant.—Whilst it seems to be established that cow's colostrum transfers immune bodies from the cow to the calf, it is questionable whether human colostrum serves a similar purpose in the case of the baby. Kuttner and Ruther, in a recent investigation, have shown that the antitoxin content of the infant's blood showed no increase attributable to colostrum, and that the omission of colostrum feeding is of no significance to the infant. The difference between the calf and the human infant in this respect is apparently due to the difference in the permeability of the cow's and human placentæ to the passage of antibodies. In the cow the placenta is impermeable to these bodies, and the colostrum is therefore the main agent for the transmission of immunity from mother to offspring. The human placenta, however, is permeable to antitoxin, &c. Thus, in the case of mothers giving a negative Schick reaction for diphtheria, their new-born babies also give a negative reaction, and there is an equal concentration of diphtheria antitoxin ($\frac{1}{30}$ unit per cubic centimetre) in the maternal and fœtal bloods; whilst in the case of mothers giving a positive Schick

[1] In a paper about to be published in the *Biochemical Journal.*

reaction, their new-born babies also give a positive reaction and the two bloods contain less than $\frac{1}{30}$ unit of antitoxin per cubic centimetre. Human colostrum occasionally contains small amounts of antitoxin, but less than the maternal or fœtal bloods.

Whilst, however, the immunological properties of human colostrum are probably very slight, the fluid is, on account of its great digestibility, believed to be a useful material to accustom the infant's alimentary tract to the work of digestion. The alleged aperient action of colostrum is disputed by some authorities.

The immune bodies in the case of the calf are transmitted by the lactalbumin which is the preponderating protein in colostrum, whether human or cow's. This protein, which is practically identical with serum albumin, can pass through the intestinal wall into the blood unchanged, carrying with it into the circulation any immune bodies it may contain, in contradistinction to all other proteins which in most cases cannot pass through a healthy intestinal wall unless they are previously broken up into their various amino-acids.

(2) *Milk.*—The chemical composition of milk, even from the same woman, varies considerably, not only at different periods of lactation but also at different parts of the day, and even at different stages of emptying of the breast. Thus the "fore-milk," i.e., the first portion obtained by the baby at the beginning of suckling, is poorer in fat than the "middle milk," whilst the last portion known as "strippings," contains the highest percentage of fat.

The following analyses (Table CXXVI) give the **percentages of the different constituents in samples of milk taken at different stages of mammary evacuation** (Forster) :—

TABLE CXXVI.

	First portion	Second portion	Third portion
Fat..	1·71	2·77	5·51
Protein	1·13	0·94	0·71

As regards milk of different women, Rotch finds that the percentage of fat may vary from 2·02 to 5·16 per cent.; lactose, from 5·68 to 7·30 per cent.; protein, 1·08 to 4·17 per cent.; salts, from 0·12 to 0·21 per cent.; and water from 84·70 to 89·68 per cent.

According to Camerer and Söldner,[1] the following table (CXXVII) represents the **composition of human milk at different periods of lactation.** The last column is taken from Pröscher[2]:—

TABLE CXXVII.

Period	Protein	Fat	Sugar	Ash	Daily increase in weight of infant
5th day	2	2·8	5·4	0·34	35-40 grm.
8th ,, to 11th day ..	1·6	3·1	6·2	0·27	
20th ,, to 40th ,, ..	1·1	3·8	6·4	0·22	
70th ,, to 120th ,, ..	1·0	2·9	6·7	0·20	22 ,,
170th ,, and after ..	0·8	2·6	6·8	0·19	18 ,,

From this it is seen that as the infant gets older and growth becomes less rapid, the protein and ash in the mother's milk decrease, and as the baby's muscular activity increases so does the proportion of carbohydrates increase too. Hence the age of a wet nurse's baby should preferably be of approximately the same age as that of her foster baby. It also follows that the usually accepted idea of increasing the percentage of protein in milk mixtures with increasing age of artificially fed babies is not based upon a physiological foundation. Nature supplies the increased protein need of the infant by furnishing it with an increased quantity of mammary secretion rather than with a milk containing a higher percentage of protein.

The following may, however, be considered as the average composition: Protein, 1·5 per cent.; fat, 3·5 per cent.; lactose, 7 per cent.; ash, 0·2 per cent.; water, 87·8 per cent.

The Proteins.—These are of two kinds, viz., caseinogen, which is insoluble in water and is present to the extent of about 1 per cent., and the whey albumins, which are soluble (lactalbumin and lactoglobulin), and which are present to the extent of about 0·5 per cent. The whey proteins are identical with the albumins of the blood-serum.

The caseinogen differs from all other proteins found in nature in regard to its chemical constitution, for while all other proteins yield on digestion in the intestine only a certain number of amino-acids varying in nature with the particular protein, caseinogen contains practically all of the numerous amino-acids

[1] *Zeitschr. f. Biol.*, xxxiii, 1896 (quoted by Hutchison).
[2] *Zeitschr. f. Physiol. Chem.*, xxiv, 1897 (quoted by Hutchison).

or "building stones" required for maintenance and growth. Thus, animals fed on the protein of wheat (gliadin) or of maize (zein) soon begin to lose weight and ultimately die, because the former lacks the amino-acid lysine, and the latter contains neither lysine nor tryptophane. If, however, these amino-acids are added, the animal's weight curve soon rises. Indeed, if tryptophane alone is added without any lysine the weight curve, instead of falling, becomes horizontal, showing that tryptophane is sufficient for maintenance of life although it is inadequate for growth. Caseinogen, however, contains not only these two amino-acids in considerable proportions, but contains also all the other amino-acids, with the exception of cystine and glycocoll, yielded by other proteins. The absence of these two amino-acids is of no great importance, because lactalbumin contains a sufficient amount of cystine to provide for growth, and cystine can be manufactured by the baby out of lactose. Hence milk forms a sufficient diet, not only for maintaining life but for growth.

The characters of the other constituents will be discussed in the next chapter, when a comparison will be made between the composition of cow's and human milk.

The Influence of the Woman's Diet on the Composition of her Milk.—Experiments on animals have shown that the nature of the mother's food has very little influence on the composition of her milk. Thus, deprivation of fat does not diminish the amount of milk fat, nor does an excess of fat increase the percentage of the fat in her milk. The same applies to lactose and minerals. Clinical experience, as well as experimental observations, has shown the same to be true in the case of woman. It is quite a common experience to find a woman who is badly nourished giving a sufficient supply of good milk, whilst, on the other hand, a mother who has the best of food may produce a milk which is either insufficient in quantity or poor in quality.

Baumm and Illner[1] made systematic analysis of milks of women fed on various sorts of diet containing different amounts of fluid, nitrogen, carbohydrate, fat, salts and alcohol (two to three pints of lager beer). They found that there was practically no variation in the composition of the milk under the influence of the various diets, except in regard to the fat content which was increased by an abundant mixed diet, or by a highly nitrogenous

[1] *Samml. Klin., Vorträg (Gynäk)*, xli, 1894 (quoted by Ralph Vincent, "The Nutrition of the Infant," London, 1913, p. 36).

diet containing much cheese, eggs and meat, and was diminished by an excess of fat in the diet.

The psychology of the mother is probably of far greater moment in this respect than the nature of the food she receives, provided her diet is of the right physiological kind. It may be stated quite definitely that in the case of most healthy mothers rationally fed the quality of their milk is of the right kind, although the quantity may, for one reason or another, such as insufficient sucking power of her baby or some psychological factor, be defective. The poorest milk producer is the neurasthenic rather than the underfed woman.

Value of Chemical Analysis to Test the Quality of Milk.—A chemical analysis of a sample of milk to test its quality is utterly useless unless the sample represents a large number of different samples taken at various times of the day and at various stages of sucking, since, as we have seen, the composition of milk, especially with regard to its fat content, is liable to considerable variation. Moreover, a milk may contain all the various chemical ingredients in the right proportions, as shown by analysis, and yet may be deficient in vitamins. The only way to tell whether a milk is of the right quality, provided it is not insufficient or excessive in quantity, is by its effect on the baby. If the baby thrives the milk is good; if it wastes or suffers from colic, or other digestive symptoms, the quality is poor.

The thumb-nail test, originally described by Soranus, is still serviceable to a limited extent.

Clinical Milk Examination.—To obtain any information of value the sample must be large and must represent a mixed specimen taken at different periods of a nursing, as well as at different parts of the day.

Reaction: Amphoteric to litmus and slightly acid to phenolphthalein.

Specific Gravity, 1026 to 1036: This is taken with a small hydrometer or urinometer. A low specific gravity indicates an excess of fat or diminution of total solids.

Microscopic Appearances: The number of fat globules in a field gives a rough idea of the percentage of fat. The size of the fat globules is of no practical significance from the point of view of nutritive value. There should be no pus, blood or colostrum corpuscles, or any other extraneous matter.

The Determination of Fat: For accurate analysis special apparatus are required, but an approximate method sufficient for clinical purposes is to allow milk to stand in a closed glass cylinder at room temperature for twenty-four hours and measure the amount of cream that has risen to the surface.

Sugar: This cannot be estimated properly without a proper quantitative chemical analysis. As, however, the percentage of this ingredient varies very little its exact determination is of little importance.

Protein: This again cannot be properly estimated without a proper quantitative chemical analysis (i.e., determination of nitrogen by Kjeldahl's method, multiplying by 6·25 and making an allowance of about 17 to 20 per cent. for non-protein nitrogen). For clinical purposes, one can form a rough idea of the amount of protein from the specific gravity and the proportion of fat—if we regard the sugar and salts as constant—so as not to affect the specific gravity. A high specific gravity will then indicate a high protein percentage, and vice versa.

Test Feeds.—But whilst the quality of a milk cannot be ascertained by chemical analysis, it is quite easy to ascertain whether the breasts secrete a sufficient quantity of milk. This is done by means of *test feeds.* After the usual interval between two feeds, the baby is weighed before and after a feed without changing its clothes or diaper in the interval. The difference in weight represents the amount of milk received by the baby. It must be remembered, however, that one test feed is not sufficient to draw any conclusions from, since the amount of milk secreted is not necessarily constant. It is necessary, therefore, to carry out this test with every feed during the day in order to ascertain the total quantity taken by the baby during the whole of the twenty-four hours.

Other indications of the quantity of milk secreted by the breasts are the degree of filling up of the breasts in the interval between the nursings, the time occupied by the infant in becoming satisfied, which should not exceed twenty minutes.

Effect of Menstruation and Pregnancy upon the Composition of Milk.—Clinical experience has failed to demonstrate any marked universal influence of menstruation or pregnancy upon the quality of the milk, though the quantity is generally diminished. Chemical analysis has shown that the composition of the milk is unchanged during those periods. Indeed, in the case of cattle it is well known that pregnancy improves the supply of milk. In individual cases, however, especially in neurotic, badly-nourished, mothers, it is found that during menstruation or with the onset of pregnancy, the supply of milk becomes diminished. While, therefore, neither pregnancy nor menstruation is a cause for weaning a baby, yet in individual cases it may be found necessary during these periods to supplement the infant's food by means of cow's milk suitably prepared, or in some cases, where the sucking may tend to produce abortions or when the strain of pregnancy is too severe upon the mother, it may be necessary to wean.

Influence of Emotion, &c.—Clinical experience shows that

nervous excitement may, in some women, have a deleterious effect upon the quantity as well as the quality of the milk, so that although chemical analysis fails to show any difference in the composition, nevertheless the milk has a bad effect upon the baby, causing it colic, or diarrhœa, &c. This effect is, however, temporary, and whilst it is advisable to leave out one or two or even more breast feeds after some shock or emotion, such nervous upsets are not as a rule indications for weaning.

Effect of Exercise.—It is believed that deficient exercise in a woman with a healthy appetite is liable to increase the percentage of both protein and fat. The same applies to excessive exercise.

Excretion of Drugs by the Breast.—Many drugs taken by the mother are excreted by the breast and may pass on to the baby. These, upon which most authorities are agreed, are iodide and bromide of potassium, salicylate of sodium, the salts of mercury, belladonna, opium, urotropin, antipyrin and alcohol. As regards the last, however, it appears in the milk only after excessive imbibing by the mother, and may then cause serious illness in the baby. Hence, whilst it is certainly not true that alcohol will cause an increased secretion of milk, as is erroneously alleged by some, there is no reason for interdicting an occasional glass of wine or beer to a mother who is in the habit of taking it.

It has been found, for instance, that in women taking a couple of pints of light beer, or five glasses of port a day, not only was the composition of the milk unaltered, but it remained free from alcohol.

As regards the arsenobenzol compounds opinions are divided. Whether, however, such drugs are or are not excreted in the milk, there is no doubt that the treatment of congenitally syphilitic babies by means of salvarsan administered to their nursing mother is not adequate; the drug should, therefore, in such cases be directly administered to the baby. The same applies to mercury. As regards magnesium sulphate, clinical experience shows that a dose of this salt given to the mother will aid the evacuation of the suckling baby's bowels. Opium and atropine, or belladonna, are probably not excreted in the milk and have very little effect upon the infant, but in view of the pharmacological action of atropine or belladonna in helping to diminish milk secretion, such drugs are best avoided during lactation. Then with regard to **toxins.** It is true that these are transmitted from mother to child by means of the milk, but such

transference is in the case of bacterial toxins of very little practical significance for two reasons: (1) because the bacterial toxins circulating in the blood of an ailing mother are very small in amount, and the amount that can get to the infant is so small as to be negligible. (2) The very small quantity which may reach the infant is partly destroyed by the infant's gastric juice, and even if not so destroyed in the stomach, being foreign proteins they cannot as a rule pass through the intestinal wall before being broken up into simpler and non-toxic substances.

Hence one may say that any mild transitory febrile disorder in the mother is no cause for weaning. Indeed, in puerperal fever wards one fails to discover any rise of temperature in babies who are fed on their mother's breasts. If, however, the mother is seriously ill with some bacterial infection, the baby is to be taken away from the breast, both on account of the risk of its absorbing toxins, as well as the harm to the mother; also because the amount of milk secreted will probably be found to be too little for the baby's requirements.

The transference of bacteria themselves in the milk is according to Basch and Weleminsky[1] a thing which never occurs unless there are severe lesions in the tissues of the breast.

In the case of non-bacterial toxins, however, e.g., when a newly-delivered mother had suffered from some pregnancy toxæmia, the matter is different, for it has been shown by A. Frost[2] that the milk of such a mother is richer in toxins even than her blood, and a child given the mother's milk even two or three days after an eclamptic fit may have fatal convulsions. In cases like these, therefore, it is wise to keep the baby away from the breast for a few days, until the symptoms of toxæmia have entirely disappeared.

Antitoxin and Immune Bodies.—Diphtheria antitoxin is constantly found in the milk of immunised animals, and the Widal reaction has been obtained with milk of mothers suffering from typhoid fever, as well as with the blood of their nursing infants. This explains to some extent the immunity of nursing infants to contagious diseases in general, although it is difficult to understand how these immune bodies manage to pass unchanged through the infant's intestinal wall—except when the infant is very young.

[1] *Jahrb. f. Kinderheilk.*, xlvii, 1898, p. 105.
[2] *Arch. Ped.*, xxix, 1912, p. 55.

The Hygiene of a Lactating Mother.

(1) *Diet.*—As we have seen, it is probable that diet has, as a rule, no influence upon the quality of the milk. If, however, the mother is overfed and has not sufficient exercise there may appear an excess of protein in her milk causing the infant indigestion. In any case, as the milk is manufactured out of the mother's blood, it is necessary that her diet be sufficient to supply her own needs as well as the needs of the baby. From the following table it will be seen that a nursing mother must increase her daily ration during lactation by some 20 per cent. of the total caloric intake. Thus an average adult woman takes :—

Protein, 138 grm. ; fat, 56 grm. ; carbohydrate, 500 = 3055 calories.

An average nursling of six months takes 900 grm. of milk ; protein, 14 grm. ; fat, 32 grm., carbohydrate, 63 grm. = 605 calories.

Therefore a nursing mother must take approximately 132 grm. of protein, 88 grm. of fat and 563 grm. of carbohydrate, in addition to an extra 900 c.c. of water.

On the other hand it is most important that she should not eat more than her appetite demands. Voit noticed an increase of milk in a bitch after ingestion of meat, and other authorities found the same to be the case in the goat. Hence some recommend an excess of protein such as rice, semolina, peas, beans, meat, fish, eggs, &c., to a lactating mother. On the other hand certain other articles of food such as spices, shell-fish, &c., should be barred on account of the likelihood of their upsetting the mother's digestion with the production of toxins which may pass to the baby. Again certain other foods such as asparagus, cabbage, &c., may impart a particular flavour to the milk which may be resented by the baby. If this is found to be the case, such articles are to be avoided. The diet must, however, contain a good supply of vitamins, otherwise the vitamin content of her milk will be defective. As regards *drinks*, the following are the percentages of alcohol in various beverages : wine 5 to 6 per cent., beer 3 to 4 per cent., cider 2 to 3 per cent. Hence, provided these drinks are taken in great moderation, the danger of the alcohol affecting the baby is not considerable. Spirits and liqueurs, however, must be forbidden.

The mother must take an adequate supply of bland fluid in order to provide enough fluid for milk formation, as well as to help the evacuation of her bowels.

(2) *Mental Hygiene.*—A nursing woman must, for reasons already given, lead a calm, serene sort of existence and be protected from any unnecessary worry or anxiety. It is said that the milk of neurotic women contains an excess of protein.

(3) *Exercise.*—A reasonable amount of exercise is absolutely necessary as it tends to diminish a tendency towards an increase in protein in the milk. It must not, however, be pushed to the point of undue fatigue, as this may aggravate the tendency which moderate exercise is intended to diminish.

Constipation must be combated by means of vegetables and fruit, abdominal massage, &c., and when the need arises by taking 10 to 15 minims of liquid cascara every evening, or some other vegetable aperient which does not get excreted by the breast, or some preparation of liquid paraffin. But saline aperients should be avoided for two reasons: (1) Because they pass through the milk and may give diarrhœa to the baby; and (2) because the watery motions which they produce may deplete the milk of its water, thus producing too concentrated a milk.

Her general health must be kept good and no carious teeth should be allowed to remain unattended to. Waller has shown that bad teeth in the mother may retard the growth of her nursling.

Care of Breasts.—The nipples should be washed with plain boiled water before and after nursing and thoroughly dried. Gentle massage of the nipples with a little cocoa butter between the index finger and thumb may help to prevent cracking of the nipples. If the breasts are somewhat pendulous, it is a comfort to have them supported by means of hammocks.

The Technique of Breast Feeding.

Position of Child and Mother.—The position should be one of ease for both mother and baby. When nursing takes place whilst the mother is in the recumbent position, both she and the baby should lie on the side, with the mouth of the latter at the level of the nipple. When feeding is carried out with the mother in the sitting posture, she should sit in a low comfortable chair, or on an ordinary chair with a small footstool to elevate her knees, on which she can rest her baby. The mother should place her index and ring fingers above and below the nipple so as to keep the breast away from the baby's nose. The baby's head should be properly supported to prevent its retraction with consequent interference with breathing and swallowing.

Duration of Feed.—The length of time which the baby should stay at the breast at each feed is not a constant factor. It varies with the sucking vigour of the baby as well as with the secretory power of the breast. It is, however, not wise to leave the baby at the breast for longer than fifteen to twenty minutes, because experience has shown that by far the greatest quantity of milk is obtained by the baby during the first five to ten minutes. Thus by weighing the baby every five minutes during a feed, Feer has obtained the following results :—

Time		Amount of milk
First five minutes	.. ..	112 grm.
Second five ,,	.. ..	64 ,,
Third five ,,	.. ..	10 ,,

If the baby does not get sufficient during the first quarter of an hour—as evidenced by weighing, as well as by the vigour with which it continues to suck—it is a sure sign of insufficient milk supply. In the majority of cases ten minutes is enough. During the nursing the nipple should once or twice be withdrawn from the baby's mouth for a few moments.

Intervals between Feeds.—The frequency of feeding is a matter about which there used to be some differences of opinion amongst different authorities. It used to be taught that during the first few weeks the baby should be put to the breast every two hours during the day and twice during the night, and that the intervals were to be lengthened during the day and the number of feeds diminished during the night as the baby grew older. This was based on observations of the anatomical capacity of the stomach at different ages, As, however, the physiological capacity of the stomach is much greater than the anatomical—since X-ray examinations show that food begins to leave the infant's stomach immediately after the beginning of sucking—this practice has been given up. The modern teaching is that the intervals should be kept constant from birth onwards up to the end of infancy, and that from 9 or 10 p.m. till 6 a.m. no feed at all should be given. The only point about which there is some difference of opinion is as to whether the interval is to be three hours or four hours. The Americans favour the four-hourly interval on the ground that a long interval allows more leisure for the mother and more time for the breasts to recuperate with the result that more milk is secreted than after shorter intervals. Also, a longer interval allows the infant more time to digest its food and to sleep, so that it wakes up more vigorous

and with a sharper appetite so that it can suck more vigorously. In this country, however, the three-hourly interval is the one generally recommended. This is based on the results of experimental evidence obtained on numbers of infants from birth onwards. X-ray examination has shown that the stomach empties itself in about two hours after a breast feed, whilst the hunger contraction method (see fig. 94) has shown that typical hunger contractions occur about three hours after a feed.

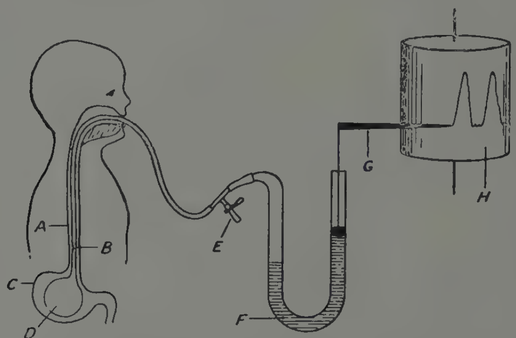

FIG. 94.—Diagram to show the method of recording hunger contraction. *A* is the child's gullet. *B*, the tube lying in it, leading to the rubber balloon *D*, which rests in the stomach *C*. *E* is the mouthpiece for inflating the balloon. *F* is the manometer, and *G* is the stylo recording on the revolving cylinder *H*. When a hunger contraction of the stomach occurs it compresses the balloon *D*, and the contraction is registered on the moving cylinder *H*. Modified from Carlson: "The Control of Hunger in Health and Disease."

Hence it is concluded that the physiological interval between feeds is between two to three hours, and three hours is the time now generally accepted. Common sense would also seem to dictate against four-hourly intervals, since on the "five feeds a day" plan it is, as a rule, impossible to give enough food at one time without overfilling the stomach.

The following is a suitable time table: 6 a.m., 9 a.m., 12 noon, 3 p.m., 6 p.m., 9 p.m.

Regularity.—Once the time table has been fixed it should be adhered to with the greatest punctuality. Irregularity not only upsets the infant's digestion and the convenience of the mother, but also interferes with the proper secretion of the milk, both as regards quantity and quality. If the baby is asleep at the time a feed is due, it must be gently awakened; in this way a habit

will be formed by the infant to wake up at the proper time. Such regularity of habit forms a very valuable training for the baby's nervous system. If the infant cries before the feed is due it should be ignored, provided it maintains a regular steady increase in weight. If, however, the crying persists regularly before or after a feed, the quantity of milk taken should be ascertained by a series of test feeds.

Alternation of Breasts.—The baby should in general be fed alternately, first on one breast then at the other, but only one breast should be given at one feed. Such an arrangement provides for complete emptying the breast, gives a long interval of rest (six hours) for each breast, and is said to insure a uniform quality of milk. If, however, the supply of milk is not adequate, it may be necessary to give both breasts at each feed, when not only will the baby get a sufficient supply, but the frequent stimulation of each breast tends to increase the secretion of milk.

Amount of Breast Milk required by the Infant.—Metabolism experiments have shown that on the average infants require about 120 calories per kilogramme body-weight (approximately 50 calories per lb.) during the first six months, in order to provide for maintenance and growth. Hence an infant weighing, say, 8 lb., will require $8 \times 50 = 400$ calories per day. Now human milk has a calorific value of about 736 calories per litre, or 20 calories per ounce. Therefore the infant will require approximately half a litre or 20 oz. of milk a day, or on the average $3\frac{1}{2}$ oz. at each feed during the first three months. A somewhat more accurate estimate can be made by measuring the surface area of the baby and allowing 1,700 calories for each square metre of surface.

Whichever method is used it must be remembered that the amount of metabolism varies with the amount of activity of the infant, the range varying almost to the extent of 100 per cent.

The following table (CXXVIII) gives the average amounts of breast milk taken by infants of different ages :—

TABLE CXXVIII.

Age	Amount of milk taken in a day	
	c.c.	oz.
1st day	15	$\frac{1}{2}$
1st week	300	10
1st month	600	20
2nd ,,	850	28
3rd ,,	850	28
4th ,,	900	30
5th ,,	950	32
6th ,,	1,000	35

Should Water be given between Feeds?—According to some authorities infants during the first three months require 3 oz. of fluid for every pound weight. As the breast infant only takes $2\frac{1}{4}$ oz. of milk per pound, it will according to them require an extra $\frac{3}{4}$ oz. of milk per pound a day, so that an 8 lb. baby will require an extra supply of 6 oz. of water a day. My own opinion is that whilst no harm as a rule results from this additional supply of water, and, indeed, during the first week it may actually help in the elimination of the excess of uric acid in the kidneys, it is in most cases unnecessary. Should it be given, however, it should be done immediately after the breast feed, since if given before a feed it may impair the appetite, and thus not only diminish the vigour of sucking with the consequent diminution of mammary secretion, but it may also result in the baby not getting enough milk at the feed. On the other hand, if given in between feeds, it is obviously going to interfere with the baby's proper amount of sleep.

Contra-indications to Maternal Nursing.—The conditions which contra-indicate breast-feeding either temporarily or permanently, may be divided into maternal and infantile:—

(A) Maternal.

Tuberculosis, whether active or latent. The former is dangerous both for mother and baby, and the latter, though safe for the baby, is a great strain upon the mother.

Infections.—Although the presence of a temporary infection is no contra-indication from the point of view of either the mother or the baby, continued infectious disease, such as severe typhoid, renders maternal nursing obviously impossible on account of the great strain upon the mother (see p. 384).

Eclampsia and Kidney Disease.—Whilst the condition persists breast-feeding is contra-indicated (see p. 384).

Epilepsy and Insanity.—If the fits are few and far between, the infant may be breast-fed under supervision. If the attacks are frequent, or if the mother is mentally unbalanced, the possibility of the mother injuring the child during a convulsion or while mentally unbalanced is a definite contra-indication.

Highly-strung women, who do not show a keen desire to nurse, should not be induced to do so.

Syphilis.—When acquired since the birth of the child syphilis is a definite contra-indication. If, however, the condition existed throughout pregnancy, not only is the disease not a contra-indication—but it is a very good reason for keeping it on the

mother's breast on the following grounds: (1) By **Profeta's** law (1865), no baby born of a syphilitic mother—even if it does not show any signs of syphilis itself—can acquire the disease from its mother's milk; Profeta's law holds good when the mother acquired her syphilis either before conception or before the fifth or sixth month of pregnancy, because in such cases the infant even when free from symptoms has a positive Wassermann reaction showing it to be in reality syphilitic. If, however, the infection occurred after the seventh month the fœtus has probably escaped contamination, and should not be nursed by the mother if its blood gives a negative Wassermann reaction. In practice, therefore, if the mother shows active syphilitic lesions, and the infant is healthy and has a negative Wassermann reaction, it is not advisable to allow the mother to suckle it. (2) such a baby may convey the disease to a wet-nurse; (3) congenitally syphilitic infants must have human milk—because on account of their debility they cannot tolerate milk of other animals so well. (4) By **Colles'** law (1837), a syphilitic infant will infect a wet nurse but not its own mother—although the latter is apparently free from disease—because it has been shown in recent years that such mothers, although they show no symptoms of the disease, give a positive Wassermann reaction. Exceptions have been published to Colles' law, and therefore in practice, if the mother's blood gives a negative reaction, she should not be allowed to suckle her infant who possesses syphilitic lesions.

Malignant disease of the breast is a definite contra-indication. Malignant disease in other parts of the body is not in itself a contra-indication from the point of view of the infant—if the mother's strength is equal to the strain.

Anæmia.—If very severe—especially after brisk hæmorrhage, nursing should be stopped. On the other hand many anæmic women improve during lactation.

Menstruation and Pregnancy.—These conditions have been discussed on p. 382.

Heart Disease.—This depends upon the severity. If the heart lesion is well compensated and the woman is eager to nurse, there is no sufficient reason for stopping her from doing so.

Conditions of the Breast.—None of the conditions occurring in the breasts are absolute bars to nursing—although some of them may render the practice very difficult. Flat nipples and fissures of the nipples interfere with nursing, but the difficulty can be overcome by means of a nipple-shield. Inflammation or

abscess of the breast are not indications for weaning, since such conditions are usually confined to the stroma of the breast and do not affect the parenchyma or milk-secreting portion—except in very late stages. The author has never found any ill effect to the baby sucking an inflamed breast. There are no abnormal constituents or toxins in the milk, and even if a few pus cells find their way into it, they do not affect the baby deleteriously. If the abscess has opened into the milk ducts and pus is obviously

FIG. 95.—Right breast : Usual nipple shield. Left breast : "Infantibus." (von Reuss.)

coming out of the nipple—nursing should be stopped. If the abscess has been opened or is discharging near the nipple—it is again an obvious indication for temporary weaning. As far as the mother is concerned, at any rate, the emptying of the breast by the baby is a great advantage—in that it prevents mammary engorgement.

(B) Infantile.

Malformation of the Mouth. Hare-lip: If the gums and palate are normal, the interference with sucking may be so slight

as to be no contra-indication to nursing. Cleft-palate, if partial, may not interfere with nursing to any large extent. Complete cleft-palate, however, generally renders sucking impossible.

Feeble-nursing.—If the baby for some reason is too feeble to suck, e.g., due to prematurity, &c., the flow in the breast should be accelerated by putting a vigorous infant to one breast while the feeble one is feeding at the other. In this way the difficulty can soon be overcome.

FIG. 96.—Right breast: Ball pump. Left breast: Aspiration nipple shield. (von Reuss.)

From what has been said it will be seen that the only absolute contra-indication to maternal nursing is tuberculosis in the mother. All the other conditions are merely relative ones.

Contra-indications to Weaning.—As far as possible weaning should not take place during the hot summer months, i.e., when epidemic diarrhœa is most likely to occur; also not during any illness of the child.

Weaning.—"Wean" is derived from the Anglo-Saxon word "wenan" which means to accustom. Weaning therefore means

getting a baby accustomed or reconciled to deprivation of its mother's milk.

Weaning Age.—The age at which weaning is practised varies in different parts of the world. In ancient times maternal nursing continued for two years or longer. In the Far East children are kept at the breast for many years. Thus, in China

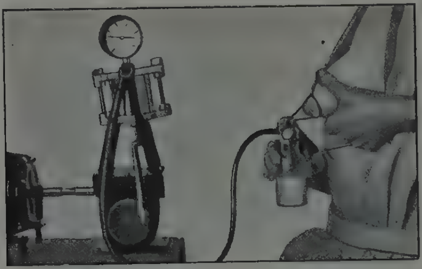

FIG. 97.—Abt's electrical breast pumps. (Edward Lasker Company New York.)

FIG. 98.—Removal of milk with Jaschne's breast pump. (von Reuss.)

and Japan for five or six years; in the Caroline Islands for ten years; and among the Esquimaux it is said that children continue to suck at the mother's breasts even at the age of 15 years. Stratz[1] records a case of a child in Java who kept on

[1] "Der Körper des Kindes," Stuttgart, 1921, p. 156.

sucking at the age of 4 years and in the intervals between sucking smoked cigarettes!

The proper age at which weaning should occur is about nine months—provided there are no contra-indications in either direction. Weaning occurring earlier is likely to give the baby digestive disturbances; whilst postponed weaning may, on account of lack of iron in the milk, give rise to anæmia in the baby.

The nutrition of the infant affords a useful guide for deciding the time of weaning. If its development is normal, its weight curve gradually rises, its flesh is firm and its colour good, then provided the mother's health and convenience do not demand weaning, breast feeding at any rate in part, may continue till the age of 12 months. If, however, the baby begins to lose weight, begins to look pale and its flesh becomes flabby, then weaning must take place at once.

Method of Weaning.—Weaning should always be practised gradually (in order to get the child's digestive organs accustomed to the change and also to prevent mammary engorgement) except in some unavoidable emergency, such as sudden dangerous illness of mother. About a month or so before complete weaning the baby should receive some bottle feeds during the day. During the first week one breast-feed during the day should be replaced by the bottle. During the second week two breast-feeds a day should be replaced. During the third week three, and during the fourth week all nursing should be replaced by the bottle. During this transition period one or more bottle-feeds of properly prepared cow's milk should be replaced by some thin cereal food. This is prepared by mixing to a smooth cream a teaspoonful of arrowroot or prepared barley with a little cold water, and then adding the amount of milk water necessary for the feed, as well as a pinch of salt, and a small piece of butter, and allowing the whole to simmer for about a quarter of an hour.

When the baby is nine months old it can as a rule quite easily cope with undiluted cow's milk.

In addition the baby ought to be given some rusk or biscuit to exercise its teeth and jaw muscles.

Signs of Under-feeding.—Under-feeding may result if either the quality or the quantity of milk secreted is at fault. As we have already seen it is usually the quantity rather than the quality that suffers. To tell that a baby is being underfed is a fairly easy matter. The only sign is that the baby does not gain

the normal amount every week. It does not as a rule cry as does a baby with colic, and it has no vomiting or diarrhœa as is the case with gastro-intestinal disturbance. On the contrary it tends rather to be constipated, the stools being small, dark-coloured, pasty, and completely digested. The urine is somewhat scanty, its reduction index (i.e., the amount of decinormal permanganate solution that can be reduced by 1 c.c. of urine) is normal (see "Child Physiology," pp. 518 and 519). Indeed, one may summarise by saying that underfeeding may be diagnosed by a stationary or falling weight curve without the symptoms of gastro-intestinal disturbance. The diagnosis is confirmed by the test feeds.

When underfeeding has been diagnosed the proper treatment is to stimulate the breasts to more efficient secretion. The mother must be given peace of mind, rest with her feet up during the day, plenty of fluid, good food and sufficient exercise. She must be encouraged in every possible way and she should have massage of the breasts after alternate applications of hot and cold compresses twice a day. The massage should be practised by placing the thumb and index finger on each side of the nipple a little behind the areola and squeezing downwards and forwards (see p. 374).

The intervals between nursing must be lengthened. This will make the baby's appetite keener and sucking more vigorous. Above all, with babies who are not too under-nourished it is advisable *not* to supplement the breast with cow's milk, so as not to appease its hunger when its sucking would be less vigorous. In this way it is possible not only to increase milk secretion, but to re-establish lactation after a few weeks' cessation.

When the milk supply is much too small and the weight curve falls rapidly, it is necessary to resort to complemental or mixed feeding.

Overfeeding.—This occurs under two conditions, either with an excessive mammary secretion even when the infant is regularly fed every three or four hours, or with a normal supply but with too short feeding intervals. The condition is diagnosed by regurgitation of part of the feed after each nursing, colic, increased number of stools. The colic is the most pronounced symptom and leads the mother to believe that her baby is hungry, with the result that the frequency of nursing is still further increased and the trouble is accentuated.

In spite of the infant taking too much food the weight soon

becomes stationary and then begins to fall. Test feeds during twenty-four hours confirm the diagnosis.

The treatment of overfeeding is proper regulation of feeding. The intervals must be not less than three hours; the duration of nursing must be reduced to a period ascertainable by means of test feeds. It may be found that the infant gets a sufficient quantity in two or three minutes. If this is done the residual milk must be removed by means of the breast pump to prevent mammary engorgement.

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CHAPTER XV

WET NURSING

"If Health and Strength permit thee, don't refuse
The Child thy nipple; nor another's use;

. . . .

Il, will the Business by that Nurse be done,
Who for another's Child neglects her own.
Yet, if thou'rt sickly, if thy spirits fail,
If the Child's touch'd with any catching Ail,
This Duty, whether hated or desir'd,
Ceases, and 'tis no more requir'd."

SCEVOLE DE ST. MARTHE (sixteenth century).

THE use of wet-nurses when mother's milk failed is mentioned in the literature throughout all ages (see Chapter II). As we have seen (p. 34), Soranus of Ephesus, in the second century A.D., gives recommendations for choosing a wet-nurse which cannot well be improved at the present day. Most writers of antiquity recognised the danger of transmission of infection from wet-nurse to infant. Some of the ancient as well as mediæval writers, however, were erroneously of opinion that the disposition of the infant is affected through the milk by the character of the foster-mother's.

Thus, Claude Quillet of the seventeenth century puts such dangers in verse as follows:—

"Oft of a venal Pap they suck their Bane
And in their Blood the Latent Plague retain.

. . . .

The Mind's affected by corrupted juice,
If bad the Milk, the Manners may be loose,
Who knows not that a Whore's malignant Pap
Corrupts the Infant in her wanton Lap;
With Lust and impious Fires it fills the Breast
And seldom is the Child, so suckled, Chast."

In the selection of a wet-nurse, attention must be paid to the following points:—

Age: about 30. Since very young mothers, or elderly ones, are apt to have a milk containing a low percentage of fat.

General Health.—She should look healthy and be free from tuberculosis (open or latent) and syphilis (as ascertained by a Wassermann test).

Development of the Breasts.—She must have well-developed breasts, but it must be remembered that the size of the breast is no criterion of its secretory capacity. The secretory power of the breasts can be ascertained from their degree of fullness and hardness three hours after nursing. The nipples must also be of the proper shape.

Her disposition must be somewhat phlegmatic, as neurotic, highly-strung women do not secrete either sufficient or good quality milk.

Her moral character should, for various reasons, be good, although it is certain that moral qualities are not transmitted by the milk.

Age of her Child.—Her child should preferably be of approximately the same age as that of the child she is given to nurse; this, however, is not an essential condition, since after the first month the milk remains of nearly constant composition.

Character of her Milk.—Her milk must be of good quality. One way of ascertaining it is by a chemical examination (see p. 381), but the best test of the quality and quantity of her milk is the condition of her own baby. If her baby is of the proper weight for its age and is happy and contented, then her milk is good. If her baby is dead, then chemical analysis of her milk is the only method for testing its quality.

When a wet-nurse has been employed, one must watch the progress of the child for a week or two; if after that period there is still no increase in the baby's weight, the nurse ought to be changed. The nurse's diet should be as nearly as possible of the kind to which she has been accustomed, as richer and more abundant foods may cause her indigestion and be deleteriously reflected in her milk.

The Provision of Human Milk without a Wet-nurse.—The employment of a wet-nurse is often a matter outside practical domestic politics. They are expensive, and are often hard to manage. As a practical alternative to wet-nursing, the experiment has recently been tried of buying milk from mothers who are willing to sell it, and supplying it in bottles to the babies in need of it. In certain children's hospitals, both in this country and America, this has been tried with considerable success. The mixed milk from a number of women is collected into bottles and dispensed in the same way as cow's milk, at a reasonable price. Premature or other weakly babies derive a great deal of benefit, not only when such human milk forms their sole diet, but even when it supplements cow's milk. The credit for this experiment is due to Dr. Franz Talbot, who started it in Boston in 1910.

CHAPTER XVI

THE ARTIFICIAL FEEDING OF INFANTS

> "Once exil'd from your breast, and doom'd to bring
> His daily nurture from a stranger spring,
> Ah, who can tell the dangers that await
> Your infant, thus abandoned to his fate?"
>
> LUIGI TANSILLO (sixteenth century).

WHILST there is no perfect substitute for human milk for infants, milk of other animals—in this country principally of the cow—is the next best thing when breast feeding is for some reason impossible. It is therefore necessary to have an exact knowledge of the differences between human and cow's milk, in order to understand the rational methods of modifying the latter to make it resemble the former, as far as it is possible to do so.

Milk, from whichever animal it comes, is a perfect food in the sense that it contains all the various ingredients required both for maintenance and growth. It is, however, a perfect food only for the particular animal for which it was intended by nature. The milks of different animals differ in their quantitative compositions and certain other characters, and it is a truism to say that nature has supplied every animal with a mammary secretion particularly suited for the specific needs of its own young (see fig. 99). We thus find that those animals which grow rapidly produce a milk which is richer in proteins and salts than the milk of an animal that grows less rapidly. Thus, to take a specific example, the young rabbit doubles its birth weight in 6 days, and the milk of its mother contains about 12 per cent. of protein and 2·50 per cent. of minerals; the calf doubles its birth weight in 47 days, and cow's milk contains 3·5 per cent. of protein and 0·72 per cent. of minerals, whilst the human infant doubles its birth weight in 180 days, and we find that human milk contains only 1·5 per cent. of protein and 0·2 per cent. of ash.

Similarly, animals which are exposed to great cold produce a milk containing a much higher percentage of fat and of higher total calorific value than those not so exposed. Thus, the milk of the dolphin contains 46 per cent. of fat, whilst that of the ass, whose young is protected against cold by a thick warm coat, contains no more than 1·4 per cent. of fat.

Further, as regards carbohydrates, those animals that are very active during the early days of life secrete a milk containing a higher percentage of sugar than those whose young are not so active. Thus, the young foal which is very energetic is provided by its mother with a milk containing about 6 per cent. of sugar,

FIG. 99.—Milk is a perfect food for the animal for which it is intended.

whilst the guinea-pig secretes a milk containing about 1·3 per cent. of sugar. Similarly, human milk contains more lactose as lactation progresses and the infant becomes more active (see further p. 379 and p. 404).

The following table (CXXIX) illustrates the quantitative differences between the milks of different species of animals used in different parts of the world:—

TABLE CXXIX.

Animal	Caseinogen	Albumin	Total proteins	Fat	Carbohydrate	Ash	Period in days during which the new-born animal doubles its weight
Woman ..	1·0	0·5	1·5	3·5	7·0	0·20	180
Mare ..	1·3	0·8	2·1	1·1	5·9	0·36	60
Cow ..	3·0	0·5	3·5	4·0	5·0	0·72	47
Goat ..	2·9	0·9	3·8	4·2	4·6	0·85	19
Sheep ..	4·2	1·0	5·2	6·2	4·2	0·93	10
Ass ..	0·8	1·1	1·9	1·4	6·2	0·47	..
Dolphin ..	..	..	17·0	46·0	1·30	0·6	..
Rabbit ..	..	..	12·0	13·5	2·0	2.50	6
Blue whale ..	..	..	12·4	20·0	5·6	1·5	..

Variability in the Composition of Milk.—The table we have given of the quantitative chemical composition of cow's milk represents an average only. The composition of milk, however, is liable to numerous natural variations, especially with regard to the proportion of fat and proteins.

(1) *Breed of Cow.*—Jersey and Guernsey cows yield a milk of high fat percentage. Holstein-Friesians produce a milk very poor in fat. The milk of Ayrshires, Shorthorns and Devons give a milk which is intermediate between the other two kinds. The variations are between 3·3 per cent. and 5·8 per cent. of fat. Similar but less marked variation is shown with respect to caseinogen, but the amount of lactalbumin is practically constant in all breeds.

(2) *Individual Differences.*—(*a*) *Morning and evening milks* have not the same composition; (*b*) *stage of mammary evacuation*: As in the case of human milk, fore-milk is very poor, and strippings are very rich in fat. Middle milk occupies an intermediate position. Hence a good milker who can empty the udder will get a milk richer in fat than another not so skilled.

(3) *Seasonal Variations.*—The effect of the season of the year upon the composition of a cow's milk has not been definitely settled.

(4) *Psychological Variations.*—A fright will alter the quantity or composition of the cow's milk, and a milker who treats a cow kindly will obtain a more plentiful supply and better quality of milk than one to whom the cow is unaccustomed.

(5) *Influence of Diet.*—All experiments go to show that apart from the influence of vitamins in the diet upon the vitamin

content of the milk, no variation in the relative proportions of the various food principles in the diet will affect the composition of the milk.

Changes which Milk may Undergo after Leaving the Cow :—

(1) *Spontaneous Changes.*—Normal milk contains carbon-dioxide loosely combined. On standing there is an exchange of gases with the air. Oxygen and nitrogen are absorbed and CO_2 is given off; the milk also absorbs any odour from its surroundings.

(2) *Effect of Heat.*—Lactalbumin is precipitated; casein is altered so as to become less coagulable by rennin; fat globules coalesce; lactose is not affected by ordinary heating but caramelises at 120° C.; gases escape, and therefore the specific gravity of the milk is raised; its acidity is diminished on account of the escape of the CO_2; the cooked taste produced is due to some decomposition of the protein with the liberation of H_2S. Organisms, but not spores, are destroyed.

(3) *Effect of Centrifugation.*—Milk is frequently centrifuged for the removal of cream. Such treatment throws the heavier solids to the bottom and sends the cream to the top.

Tocher[1] gives a number of correlation coefficients between two variables, in the case of cows of all breeds, from which the following Table CXXX has been constructed :—

TABLE CXXX.

Variables	Correlation coefficient	Remarks
Age and fat	$-\cdot0430 \pm \cdot026$	Not significant
Age and yield	$+\cdot0767 \pm \cdot026$	„ „
Age and total nitrogen	$-\cdot0329 \pm \cdot026$	„ „
Age and lactose	$-\cdot1693 \pm \cdot025$	Small but significant
Lactose and fat	$-\cdot0577 \pm \cdot026$	Not significant
Ash and total nitrogen	$+\cdot4566 \pm \cdot021$	Significant
Ash and casein nitrogen	$+\cdot4190 \pm \cdot026$	„
Ash and albumin nitrogen	$+\cdot1681 \pm \cdot030$	Small but significant

Differences between Human and Cow's Milk.

(1) *Quantitative Composition.*—If we summarise what we have stated above with regard to the two kinds of milk, we get the following table :—

TABLE CXXXI.

Milk	Water	Caseinogen	Albumin	Fat	Carbohydrate	Salts
Human	86·4	7·0	0·5	3·5	7·0	0·2
Cow's	86·0	3·0	0·5	5·5	5·0	0·7

[1] J. F. Tocher, "Variations in the Composition of Milk," Edinburgh, 1925.

The relatively low percentage of protein and ash in human milk is, as we have seen, accounted for by the comparatively slow growth of the human infant, but in view of the relatively smaller amount of muscular activity of the infant as compared with the calf, the greater percentage of carbohydrate in human milk is somewhat surprising. It is possible, however, that the greater rate of myelination of the infant's brain-fibres requires more carbohydrate, since that substance enters into the composition of galactolysin which is a constituent of myelin.

The author, from a study of the nature of the plantar reflex in early life, found that before the age of 1 month there was a higher percentage of cases of the extensor (or dorsiflexor) type of response (Babinski phenomenon) in bottle-fed than in breast-fed babies, suggesting that the deficiency of lactose in the cow's milk delayed the myelination of the pyramidal tracts upon the development of which the nature of the plantar reflex depends.[1]

(2) *Qualitative Composition.*—Both kinds of protein, though very nearly respectively equal in composition in both kinds of milk, are not exactly identical. They differ in certain immunological properties. Thus, when animals receive repeated injections of cow's milk, their blood-serum will precipitate the proteins in cow's milk but not those of human milk. *Per contra*, the blood of animals similarly sensitised with human milk will precipitate human but not cow's milk proteins, showing that the proteins of the two kinds of milk are biologically different. The same applies to the proteins of milk from other animals. Indeed, by such immunological methods it is quite easy to differentiate between milks of animals of different species. This is a matter of practical importance in cases where human milk is bought at high prices for the feeding of delicate infants.

It has further been shown by Dudley and Woodman[2] that the amino-acids derived from different caseinogens have different intramolecular structures, as shown by their optical activities.

(i) **Caseinogen** is a nucleo-albumin which contains phosphorus, is insoluble in water, moderately soluble in alkalies, and precipitated by acids and rennin. It is not coagulated by boiling. Human caseinogen yields different proportions of the various amino-acids, is said to contain less phosphorus and requires a higher acidity for its precipitation by rennin than does cow's caseinogen. Thus, while bovine caseinogen will clot with rennin

[1] Feldman, W. M. "The Nature of the Plantar Reflex in Early Life and the Causes of its Variation." *Amer. Journ. Dis. of Children*, xxxii, 1922.

[2] *Bioch. Journ.*, ix, 1915, p. 98.

under an acidity which is expressed in hydrogen-ion concentration as $C_H = 1{\cdot}1 \times 10^{-6}$, human caseinogen requires one of 3×10^{-7}. Moreover, the precipitate consists of fine floury curds which can easily leave the stomach, in the case of the human caseinogen, but of tough and heavy masses which stick to the stomach and cannot readily leave it in the case of bovine caseinogen. This difference in the behaviour of the two kinds of caseinogen seems to be correlated with the peculiarities of the digestive tracts in the two species. In the cow the digestive surface of the stomach forms about 70 per cent. of the entire alimentary tract, and the digestion of protein is mainly gastric. Hence it is necessary for the curds to be tough in order that they may not readily escape. In the human infant, however, the stomach forms only about 20 per cent. of the total digestive surface, and while digestion partly begins in the stomach, it is mostly carried on in the intestine, and the fineness of the curd renders its escape from the stomach into the intestine easy.

The caseinogen is present in combination with calcium as soluble calcium caseinate. It is never found in any other substance than milk, not even in the body of the animals producing the milk, with the exception of the sebaceous glands, of which the mammary glands are modified structures.

(ii) **Lactalbumin**, which is also never found anywhere else in nature, is almost identical with serum albumin, but differs from it in its light rotatory power. In contradistinction to caseinogen it is coagulated by heat. It is believed that it is the very small amount of lactoglobulin in the milk which carries the immune bodies (see further, Chapter XIV, p. 378).

Human lactalbumin is richer in lysin and tryptophane (Leonard Findlay).

Milk also contains lactoglobulin, but its quantity is very small. The lactalbumin and lactoglobulin are both together called the whey proteins.

Factors which Influence the Character of the Casein Curd :—

(1) *Dilution with water* not only reduces the proportion of caseinogen and thus renders the clot thinner on account of the smaller concentration, but it also makes the curds in the stomach smaller, softer and more friable, because it reduces the amount of soluble calcium salts on which coagulation depends. Indeed, the addition of $CaCl_2$ to diluted milk will hasten the coagulation.

(2) *Dilution with cereal waters*, such as barley water, renders the curds smaller and softer, because the colloidal substance of

the slimy cereal waters acts like mucus in preventing the agglutination of smaller curds into larger masses.

(3) *Boiling* has a great effect on the prevention of tough curd formation. Pasteurisation has no effect on coagulation.

(4) *Alkalies*, e.g., lime water, bicarbonate of soda, &c., cause the milk to be coagulated in the stomach in the form of small, thin, and friable clots. The effect upon the curd is in direct proportion to the amount of alkali used. The amount commonly used is 1 oz. of lime water to every pint of milk (i.e., 5 per cent.), but larger amounts may be used. The action is probably due to the neutralisation of gastric HCl.

(5) *Sodium citrate*, in the proportion of 2 gr. to the ounce of milk, has an effect similar to that produced by alkalies.

The action is as follows :—

(i) One molecule of calcium caseinate (soluble caseinogen) + rennin = 2 molecules of calcium paracaseinate (insoluble caseinogen).

(ii) Calcium caseinate + sodium citrate = sodium caseinate + calcium citrate.

(iii) Sodium caseinate + rennin = 2 sodium paracaseinate (soluble).

[About 1·7 gr. of sodium citrate to 1 oz. of milk prevents coagulation.]

(6) *Peptonisation.*—This of course converts the caseinogen into peptone, which is not coagulable. Peptonisation when carried out by means of Fairchild's peptogenic milk powder, which in addition to pepsin contains also some bicarbonate of soda which renders the milk alkaline, produces milk which is said to be almost identical in composition with human milk.

(7) *Dried or Condensed Milk.*—The clot formed in these cases is very small and flocculent.

(8) *Precoagulation.*—This is an ingenious method of insuring a small flocculent precipitate of casein in the stomach. The milk is previously treated with rennin to coagulate the casein. The clot is then finely broken up by shaking or stirring, and allowed to stand. This fine, broken-up clot does not afterwards recoagulate in the stomach.[1]

Fat.—The fat of human milk has a lower melting point than has that of cow's milk, because it contains a higher percentage of oleic acid, i.e., 50 per cent., as compared with 34 per cent. in the case of cow's milk. Its fat globules are also smaller, a fact which is believed to make it more digestible than cow's milk. On the other hand, the fat of human milk is relatively poor in volatile fatty acids, such as butyric and caproic, the percentages

[1] V. Dungern, *Münch. med. Wochenschr.*, xlvii, 1900.

in the milks being 2·5 per cent. and 2·7 per cent. respectively. Amongst the fats there are the *lecithins* to be considered. Lecithin is a phosphorised fat in combination with a nitrogenous basic radicle, and is found in brain and nerve tissue. Human milk contains, in proportion to protein, a higher percentage of lecithin than does cow's milk. This is to be expected if we consider the relative growth of the human and calf's brain. Milk fat, like casein, is not found anywhere else than in milk, and the yellowish tinge of cow's milk is due to the carotin or xanthophyll in the cow's food colouring the fat globules.

(iii) **Lactose.**—This seems to be identical in composition and character in the milks of different species, including woman. It is a disaccharide composed of dextrose and galactose. As the myelin of nerve fibres contains galactose, it is possible that the higher percentage of lactose in human milk is a provision of nature for the rapid myelination of the brain and nerve fibres occurring in the first few weeks of post-natal life of the human infant.[1] From this point of view, therefore, cane sugar is not a proper substitute for lactose in the artificial feeding of infants. Lactose is never found anywhere else in nature except sometimes in the urine of the lactating mother. It is less soluble in water than other sugars and is therefore not so sweet.

(iv) **The Salts.**—Cow's milk contains about three and half times as much of salts as does human milk. Indeed, there is about the same relation between the proportion of total minerals as between that of total proteins in the two kinds of milk. The relative amounts of individual salts, however, is not necessarily the same. Thus, calcium, in virtue of its combination with caseinogen, is present in proportion of about 1 : 1; on the other hand, potassium and iron salts are present in relatively smaller proportion in the two milks. Phosphorus is also present in greater proportion than 3 : 1 in the two milks.

(v) **Bacteria.**—Human milk drawn by the infant is commonly said to be sterile. This, however, is not literally true, since a few stray organisms, such as the *Staphylococcus aureus*, find their way into milk ducts through the nipple and thus contaminate the fore-milk. They are, however, invariably non-pathogenic and innocuous. A few septic organisms also may find their way into the milk in cases of mastitis, as well as from unclean nipples. These again are very few and harmless, as a rule, when swallowed.

[1] See W. M. Feldman, "The Nature of the Plantar Reflex in Early Life," *Amer. Journ. Dis. Childr.*, xxiii, 1922, pp. 23 and 28.

Cow's milk has a very high bacterial content, for although inside the udder of a *healthy* cow it is either practically sterile or contains the same kind and number of organisms as human milk, yet it is liable to contamination from numerous sources before it reaches the infant. These external sources are:—

Contamination from the Udder.—Micrococci, streptococci, *Bacillus coli*, and, in the case of a tuberculous udder, the tubercle bacillus (figs. 101-105).

Contamination during Milking.—(1) From the coat of the cow; (2) dust from the air; (3) from the hands of the milker. The milker may introduce pathogenic bacteria by being a carrier of typhoid, diphtheria and tubercle bacilli, as well as of the virus of scarlet fever.

Contamination from utensils, e.g., pails, cans, &c. Contamination at the hands of the distributors.

The illustration (fig. 100) gives a pictorial representation of the numerous sources of milk contamination before it reaches the baby.

Dr. Thomas Orr, in 1908, made an examination on behalf of various Yorkshire authorities and found that for 100 organisms found in milk the following sources were responsible to the extent stated:—

Farm	41·4 per cent.
Railway transport	21·1 ,,
Dairy	18·5 ,,
Consumer's house	19·0 ,,
Total ..	100 per cent.

The number of bacteria in milk cannot be given accurately, since it varies within wide limits. London milk may, however, be said to contain about $3\frac{1}{2}$ million per cubic centimetre. This number would give quite a cloudy appearance to a transparent liquid like water, but in milk they are invisible on account of its opacity. A rough estimate may, however, be made from the amount of sediment.

Of the numerous bacteria found in cow's milk, a large number are not only harmless but beneficial, since they include many varieties of the lactic acid-producing organisms (probably more than a hundred, the commonest of which are the *Bacillus acidi lactici* and the *Streptococcus lacticus*), which antagonise the action of certain putrefactive germs. Indeed, some physicians use buttermilk made from soured milk in certain digestive disturbances of infancy (in which there is reason to believe that there is a preponderance of proteolytic over lactic acid organisms in the infant's


The Long
versus
The Short Haul
A great number of babies get their food through a tube many miles in length.
It often takes a day and a half for the milk to run from the cow end of the tube to the baby end of the tube.
This tube is open in many places and baby's food is frequently polluted. It is often wrongly kept in over-heated places.
There may be a diseased cow at the country end of the tube.
AND YET SOME PEOPLE WONDER WHY SO MANY BABIES DIE!
On the other hand the mother-fed baby gets its milk fresh, pure and wholesome—no germs can get into it.
TO LESSEN BABY DEATHS LET US HAVE MORE MOTHER-FED BABIES.
You can't improve on Nature's Plan.
FOR YOUR BABY'S SAKE NURSE IT.

FIG. 100.

intestines) with considerable success. The author has himself used such milk fairly extensively during a considerable number of years at the Infants Hospital with some success.

But while the vast majority (about 95 per cent.) of the organisms are harmless or beneficial, there are a number of pathogenic organisms, such as the tubercle bacillus, the diphtheria bacillus, the scarlet fever organism, the typhoid bacillus, the organisms of epidemic diarrhœa, &c., which are of enormous importance from the point of view of infant hygiene. Of these the most important one is the tubercle bacillus. This organism finds entry into the milk either directly from the infected cow (bovine bacillus), or infected milkman (human bacillus), or indirectly from the tuberculous cow. The indirect contamination of the milk takes place from the manure containing fæces in which are present multitudes of live tubercle bacilli that have reached it from tuberculous sputum swallowed by the cow from an open focus in its own lungs.[1] It is believed that at least 25 per cent. of all dairy cows are affected with tuberculosis. In addition the milk may contain putrefactive organisms, which in most cases are kept in check by the antagonistic action of the lactic acid organisms, unless the latter are either destroyed by heating (which, however, does not kill the spores of the putrefactive organisms) or have been thrown out of action as the result of the total using up of the lactose, when the putrefactive organisms may flourish.

Milk and Tuberculosis.—The relationship between tuberculosis in cows and human tuberculosis is a matter of great theoretical and practical interest and importance. According to some authorities, the ingestion of cow's milk containing bovine tubercle bacilli helps the baby to become immunised against the human type of the disease. On the other hand, it has been estimated by others that, in addition to 3,000 deaths from tuberculosis per year originating from cow's milk, tens of thousands of children are crippled or otherwise incapacitated by various forms of surgical tuberculosis. Dr. A. Stanley Griffith[2] finds that out of 1,450 cases of all sorts of tuberculosis at different ages, there were 125 cases of cervical gland tuberculosis, of which 65 were of human and 60 of bovine origin, i.e., a percentage of 48 of bovine tuberculosis. Of 514 cases of bone and joint tuberculosis, 99

[1] Report of Medical Research Council, 1920, quoted by Lane-Claypon.

[2] *Proc. Roy. Soc. Med.*, Sept., 1925, vol. xviii (Sections of Epidemiology, Comp. Med., and Dis. of Children), p. 88.

(i.e., 19·2 per cent.) were of bovine origin. Of 52 cases of skin tuberculosis, 20 (i.e., 38·4 per cent.) were of bovine origin, and so on.

Classifying his cases according to age incidence, his findings agreed with the observations of previous authorities, that in all the forms of tuberculosis the percentage of the bovine type was highest in children under 5, and it gradually diminished with increase of age. Thus, amongst the cases of cervical gland tuberculosis there was an incidence of 85 per cent. bovine in the age period 0 to 5 years, and of 18·2 per cent. at 20 years and upwards.

TABLE CXXXII.

PERCENTAGE INCIDENCE OF BOVINE TUBERCULOSIS OF DIFFERENT PARTS OF THE BODY IN CHILDREN UNDER 5 YEARS AND AT ALL AGES.

Tuberculosis	Under 5	5 years and upwards	All ages
Cervical glands	85·0 per cent.	41 per cent.	48 per cent.
Bones and joints	30·2 ,,	17 ,,	19·2 ,,
Skin abscesses (T.B.)	58·3 ,,	31 ,,	38 4 ,,
Lupus	66 0 ,,	43 ,,	51·4 ,,
Brain	4·6 ,,	Less	Less
Abdomen	58·0 ,,	Less	Less

Further, post-mortem examination showed that in 80 per cent. of all cases of tuberculosis in children under 12 years of age the portal of entry was the alimentary tract, showing that milk was the cause of infection.

It is also estimated that 1,000,000 dairy cows (out of a total of 3,000,000) in this country are tuberculous, and of these 5 per cent., or 150,000, are infected in the udder, rendering the milk highly dangerous (Nathan Raw). If we remember that on the average each cow provides no less than 1,500 gallons of milk a year, a large proportion of which is drunk by infants, the importance of the subject of milk and tuberculosis will be realised. Samples of milk examined in different parts of the country, as well as in different parts of the world, showed the presence of tubercle bacilli, as proved by the inoculation of guinea-pigs, in percentages ranging between 7·3 per cent. (London) and 20 per cent. (Edinburgh).

As regards the transmission of *human* tuberculosis through the milk (i.e., from the expectoration of a tuberculous milker, &c.), it is probable that the danger is not so great as is the transmission of the bovine type.

So much for one side of the picture. Now what about the other side? In the first instance, von Pirquet, the eminent Austrian authority, states[1] that practically all the cases of surgical tuberculosis in children in Vienna are of human origin. Secondly, many authorities believe that the frequent taking of small doses of tubercle bacilli confers an immunity against the disease, and they assert that if bovine tuberculosis would be eliminated a race of children would grow up that would present a virgin soil in later life.

Dr. S. T. Irwin, in a letter to the *British Medical Journal* in 1926, records a case where a child, aged 2—who had been always fed on tubercle-free milk (obtained from tuberculin tested cows)—until he went to a place where only ordinary milk was obtainable; within seven weeks he developed glands in the neck which contained tubercle bacilli. He makes the plausible suggestion that the previous feeding with tubercle-free milk may have rendered the child specially susceptible to the onslaught of the tubercle bacillus in doses of such moderate size as would not be harmful to other children who had hitherto acquired some immunity to the disease by being fed on ordinary milk.

On which side does the truth lie? The answer seems to be that, so far as Pirquet's contention is concerned, it can be easily reconciled with the results found by Griffith, Frazer, and others, since Pirquet states that the whole of the milk in Vienna is boiled. As regards the possible immunisation against human tubercle by ingesting small doses of the bovine bacillus, it is to be remarked that while there is very little doubt, from the work of Calmette and his co-workers, that immunisation may take place in this way, it is not quite a scientific procedure to give infants unmeasured doses of tubercle bacilli in the hope that they may be small enough to confer immunity. What seems to be more logical is to feed infants on milk known to be free from tubercle bacilli, and keep them away from all other sources of possible infection, but in order to avoid the risk of rearing a population susceptible to tuberculosis, an attempt should be made, if possible, to protect them permanently by methods of passive immunisation (see Chapter XXIV, Section on Tuberculosis).

Milk Pasteurisation and the Non-pulmonary Tuberculous Death-rate.[2]

In New York City, in 1910, only 50 per cent. of the milk consumed was pasteurised. In 1914 pasteurisation of all milk, save that grade termed certified, was made obligatory by law. The following statistics of the non-pulmonary tuberculosis death-rate reflect the results:—

[1] Quoted by Lane-Claypon, "Milk and its Hygienic Relations," p. 256.

[2] *Medical Officer*, October 16, 1926.

TABLE CXXXIII.

Years	Non-pulmonary death-rate	Years	Non-pulmonary death-rate
1910	0·29	1918	0·24
1911	0·30	1919	0·20
1912	0·28	1920	0·17
1913	0·28	1921	0·14
1914	0·27	1922	0·13
1915	0 27	1923	0·12
1916	0·23	1924	0·14
1917	0·24	1925	0·12

Moreover, whilst in the pre-pasteurisation days the cervical glands in the non-pulmonary T.B. cases contained the bovine T.B. in more than 50 per cent., in only six specimens was it found since pasteurisation. In five of these the patients came from outside New York, where they were fed on raw milk.

Diphtheria and Milk.—Amongst the various agents which convey the diphtheria bacillus, milk must be accounted as one of the most important. The milk usually gets contaminated at its source by being handled at the farm by people who are either actually suffering from or are carriers of the disease. In an outbreak of diphtheria in South London, in 1914, it was discovered that all the houses in which the cases occurred were supplied with milk from a certain Lambeth dairy, which in turn obtained its milk from seventeen farms in Sussex and Surrey. On examining each farm it was discovered that a milker on a Sussex farm was suffering from "milker's finger," which, on bacteriological examination revealed virulent diphtheria bacilli.

The cow itself may have sores on its teats containing diphtheria bacilli.

The Significance of the other Milk Organisms.—We have seen that the vast majority of the organisms found in milk are harmless. The pathogenic or disease-producing organisms, however, though insignificant in number, are of the utmost importance from the point of view of the health and welfare of the infant. But even the non-pathogenic bacteria, though harmless in small numbers, may, if present in large numbers, give rise to gastro-intestinal trouble in the infant. Thus, Park and Holt[1] fed numbers of infants on milks containing varying numbers of organisms and found the following results:—

[1] *Arch. Ped.*, xx, December, 1903, p. 88.

TABLE CXXXIV.

Kind of milk	Number of infants fed	Average weekly gain in ounces	Diarrhœa		Death
			Mild	Severe	
Pure (2 bacteria per c.c. after boiling)	41	3	10	—	1
Pure (20,000-200,000 bacteria per c.c.) heated at home	47	4	16	7	0
Ordinary (1,000,000-25,000,000 bacteria per c.c.) heated at home ..	30	4	7	8	1
Cheap (400,000-175,000,000 bacteria per c.c.)	28	$\frac{1}{4}$	5	16	4

The pathogenic germs are those responsible for streptococcal sore throats, typhoid fever, scarlet fever, diphtheria, and cholera. In addition to the actual dangers of bacteria in milk, these organisms destroy the food value of the milk by breaking up either the lactose or the proteins.

FIG. 101.—Immense wall of manure just outside cow-shed. Cows milked inside the shed.

Safe Milk.—From what has been said, we see that: (1) Some milk sold for infant use is highly contaminated with bacteria; (2) even the harmless bacteria may do mischief if present in large numbers; (3) the harmful bacteria are responsible for epidemics of different infectious diseases as well as tuberculosis. From this it follows that, to protect babies from harm, milk before it is given to an infant must be freed from organisms as far as possible. This can be done by demanding great cleanliness

FIG. 102.—The manure-caked flank of a cow.

FIG. 103.—A very old and visibly tuberculous cow.

in the collection, storage and supply of milk obtained from cows that have been certified to be free from disease (figs. 106-109).

Standardised and certified milk was first introduced in America as the result of the exertions of Dr. Henry L. Coit, in 1882.

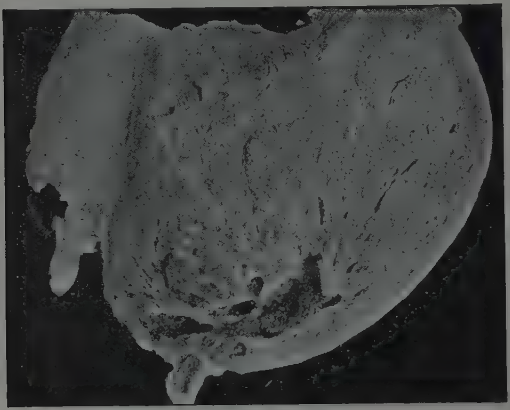

Fig. 104.—Udder of cow, showing tuberculous lesions.

Fig. 105.—An exceptionally dangerous cow. The only evidence that the cow was tuberculous was the result of the tuberculin test.

Similar legislation was passed in this country under the Milk and Dairy Act, 1922, which laid down the following three standards of purity of milk:—

(1) *Certified Milk.*—This milk is derived from cows which

have been certified as free from tuberculosis by the tuberculin test, and to be generally in a good state of health. The whole herd must pass the tuberculin test and other veterinary examina-

FIG. 106.—Concrete yard outside milking barn to prevent cows from tracking in the mud.

FIG. 107.—Interior of milking barn, Mundsmere Manor, Basingbroke.

tion every six months, and new cows admitted to the herd must undergo immediate examination. It must not contain more than 30,000 bacteria per cubic centimetre.

(2) *Grade A* (*Tuberculin Tested*).—Must answer the same conditions as the certified milk, except that the minimum bacterial content is 200,000 per cubic centimetre.

(3) *Grade A*.—Same as No. 2, except that it is not guaranteed against tuberculosis.

The Requirements of a Good Milk.

(1) The cows from which the milk comes must be free from tuberculosis, as certified by a veterinary surgeon.

(2) The cow-shed must be clean and sanitary (figs. 106 and 107).

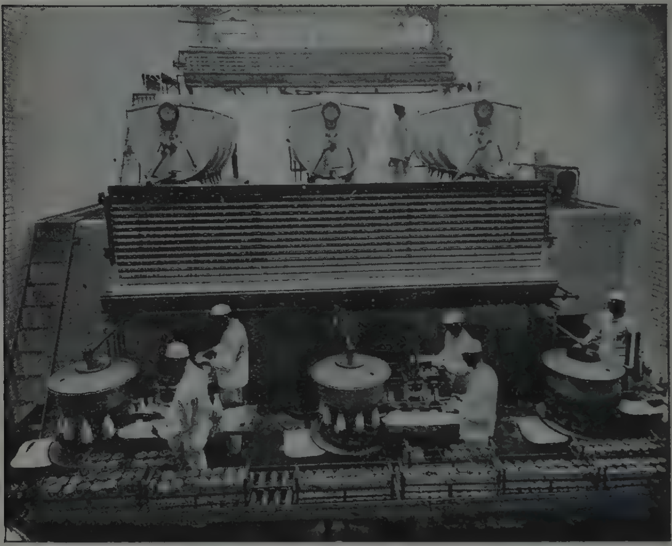

FIG. 108.—Interior of good modern dairy, where the milk is bottled and capped automatically after pasteurisation.

(3) The utensils must be clean.

(4) The milker must be free from tuberculosis or other communicable infectious disease, and his hands must be free from sores, as certified by a physician. Epidemics of scarlet fever, diphtheria and typhoid have been traced to infected milk.

(5) The milking must be done under hygienic and sanitary conditions :—

(i) The cows must be kept clean, so that no dirt comes from their hair, and their udders must be washed just before milking. It has been shown that a single hair from the cow falling into the milk may add 27,000 bacteria.

(ii) No loose hay or manure may be left in the shed during milking, as one grain of manure may contribute a thousand million living microbes. According to Dr. Backhaus, the people in Berlin consume 3 cwt. of fæcal matter a day from the manure which finds its way into milk.

(iii) The utensils must be washed with boiling water and steamed.

(iv) The milker must wash his hands with soap and water just before milking and must wear clean clothes. He must never moisten his hands for milking, either with his own saliva or with milk—a practice frequently adopted to facilitate the process.

(v) The first few streams of milk must be discarded (see p. 408).

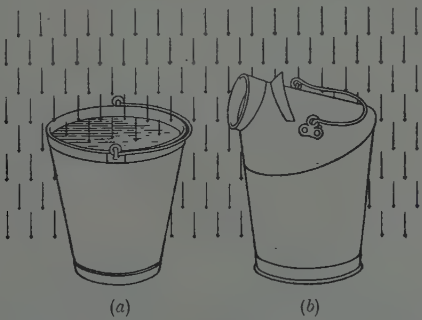

FIG. 109.—The old (*a*) and modern (*b*) milk pails compared. No dust can fall into the latter.

In the opinion of some authorities (e.g., Nathan Raw) the only way to reduce tuberculosis in cows is to immunise calves as they are born, and then annually, with attenuated living cultures of tubercle bacilli or with cultures of dead bacilli, and the Ministry of Agriculture has established a scheme for carrying this out. There is, however, a great objection to this method, viz., the immunised animals react to the tuberculin test, and it

will therefore be difficult to say whether they are naturally nfected or not.

(vi) The pail must be of the *protected* variety shown in the illustration (fig. 109). Such a utensil excludes, to a large extent, dust and dirt. Lane-Claypon gives the following bacterial counts (per cubic centimetre) in milks collected in open and protected pails respectively :—

TABLE CXXXV.

	Open pail	Protected pail
After 2 hours	41,312	234
,, 24 ,, ...	1,803,800	4,975
,, 48 ,, ...	28,790,000	1,968,375

(6) The milk must be strained at once through a sterile cloth, and must be put on ice and kept there in sealed bottles (to keep out dust and flies) until ready for consumption, since warmth increases the bacterial count.

Influence of Temperature on Bacterial Content of Milk.— The milk as soon as collected should be properly refrigerated, since even in high grade milk containing 3,000 bacteria per cubic centimetre, after twenty-four hours at 0·68° C., the count may rise to 450,000 per cubic centimetre, and after forty-eight hours the number may rise to 25,000,000 per cubic centimetre. Whilst if the original number is 30,000 per cubic centimetre, it may rise after forty-eight hours at the same temperature to 25,000,000 per cubic centimetre.

(7) The bacterial content of the milk so produced should not be higher than 10,000 to 30,000 per cubic centimetre, and should contain no pathogenic organisms, as certified by a bacteriologist. Such milk, produced under supervision, is called certified milk, because when the authorities are satisfied that it has been produced in accordance with the above requirements, they give the farmer a certificate which he is allowed to place on each bottle of milk. The authorities also guarantee that the fat content is 4 per cent., as certified by a chemist. It must be remembered, however, that although certified milk is the best that human ingenuity can devise, yet "it assumes a reliable and ever-vigilant milk commission and honest producers and carriers. Any weak point in this human chain can leave us in a fool's paradise" (Bannerman).

Indeed, certain samples of such milk have been found to contain living tubercle bacilli, and Dr. Park, of New York, stated that all of the six cases of bovine tuberculosis in infants investigated by him had been fed on certified milk.

Removal of Bacteria from Milk.—Whilst under ideal conditions milk should be so pure as not to contain any pathogenic organisms, such perfection can never be guaranteed. Hence it is necessary to subject even the best milk to such processes as will remove the pathogenic organisms. This may be done in several ways.

(1) *HEATING*:—

(*a*) *Sterilisation by Heat.*—The milk is raised to a temperature higher than 100° C. or 212° F., and kept at that temperature for a few minutes. Such treatment destroys all the bacteria, but it also destroys the milk.

(*b*) *Boiling.*—The milk is raised to 100° C. or 212° F., and kept at that temperature for a minute or two. Under this treatment all the bacteria are destroyed without spoiling the milk, but bacterial spores survive.

(*c*) *Pasteurisation* is the name applied to the method of destroying organisms by means of heat at a lower temperature than 100° C. or 212° F., when such temperature is applied for a long time. It is a process originally applied by Pasteur for the purpose of preserving wine and beer. For removing bacteria from milk, the temperature used is 145° F. (62·5° C.) for thirty minutes, or 160° F. (71° C.) for twenty minutes. Pasteurisation, like boiling, will kill all the bacteria, including the tubercle bacillus, but not the spores which come from the putrefactive organisms. Hence, whether the milk is boiled or pasteurised, it must be either used at once, or it must be kept in a refrigerator below 50° F. to prevent germination of the spores. If this is not done the spores will give rise to putrefactive organisms which, unhampered by the fermentative bacteria (lactic acid organisms) that have been killed right out, will begin to break up the protein, and not only reduce the caloric value of the milk but will also produce poisonous products that will upset the infant (although it has been recently shown by Ayers and Johnson that such milk contains a great number of lactic acid bacteria).

The Relative Merits of Boiling and Pasteurisation.—There is really very little to choose, from a practical standpoint, between the two methods. On a commercial scale pasteurisation, on account of the lower temperature employed, is cheaper. The

cost of pasteurisation on a commercial scale is about a halfpenny per six gallons. For home use, however, boiling is probably less troublesome, although on account of the frothing up of the milk and its flowing over the pan it is rather messy. Further, boiling, as we have seen (p. 406) renders the milk more digestible.

Neither boiling nor pasteurisation destroys the antirachitic vitamin, but boiling, if continued for a long time, may kill the antiscorbutic vitamin C. The effect of quick boiling on this vitamin is negligible, and clinical experience has shown that there is no relation between boiled milk and scurvy. In any case, any possible harm may be avoided by giving the baby fruit juice.

Milk should never be pasteurised at the source, but should be delivered raw in sealed bottles, because: (1) Unless every precaution is taken to keep the milk iced during transmission the spores will grow again; (2) the consumer, not knowing that it had already been pasteurised, will heat it again before using, which is undesirable.

(2) *ULTRA-VIOLET RAYS* destroy bacteria, and as irradiation also augments the antirachitic property of milk, such a method may turn out to be the ideal one, since for all other purposes a milk so treated would probably remain "raw." For the present, however, this method cannot for obvious reasons be used at home, although in hospitals and infant welfare centres it may be found to be quite practicable. On the other hand, as Bannerman wittily remarks, as raw milk produces a very hard curd, but boiled milk renders the curd soft and flocculent, it is just as reasonable to cook clean fresh milk as it is to cook a clean fresh potato or turnip.

(3) *PRESERVATION BY COLD.*—Cooling of milk to below 10° C. will check the growth of organisms but will not kill them.

(4) *PRESERVATION BY CHEMICALS.*—An ideal chemical milk preservative would be a substance possessing the following three properties, viz.: (1) Harmlessness to the consumer (i.e., the infant); (2) germicidal action on all pathogenic organisms, including spores; (3) non-interference with the chemical and biological characters of the milk. Such a preservative has not as yet been discovered. The chemical preservatives which have been most frequently added to milk are formaldehyde, boric acid, borax, benzoic acid, salicylic acid and hydrogen peroxide. These substances have a selective action; in certain proportions they inhibit the growth of the fermentative organisms without checking the growth of the pathogenic ones.

The use of all such chemical preservatives is now illegal for various reasons. Firstly, they encourage the sale of dirty milk, whose objectionable characters they conceal. Secondly, although in small doses they are harmless to the consumer, there is a temptation for the addition of preservative to the milk during the various stages of its transmission, and may thus reach a stage when they become injurious.

(5) *PRESERVATION BY DRYING.*—Bacteria contain 70 to 90 per cent. of water, and as they receive their nourishment through the cell wall their food must be soluble and dissolved in water. Hence a food which is dried cannot support the life of putrefactive organisms and therefore does not decompose. Milk dried to a powder, or which has been "condensed" by concentration and the addition of sugar, can be kept for a long time without decomposing. When milk is condensed there is enough water for the growth of the bacteria, but the addition of cane-sugar exerts an osmotic pressure which prevents the available water from passing into the bacterial cell.

The chemical preservation of milk must unreservedly be condemned on the following grounds:—

(1) Large doses of preservatives are harmful, and as the milk may be dosed by the farmer, the middleman and the actual purveyor, the amount of preservative may be very considerable.

(2) It is possible that preservatives may check the growth of the lactic acid organisms, which visibly alter the milk, without their affecting the putrefactive ones.

(3) Their addition is a direct incentive to dirty methods of milking. Good, cleanly-collected milk requires no chemical preservative.

(4) Preservatives, though harmless in small quantities, may have an injurious cumulative action.

The following tests will detect formaldehyde and boric acid in milk.

Test for Formaldehyde.—Mix equal parts of the suspected milk and commercial sulphuric acid. A purple line at the level of separation between the milk and the reagent indicates the presence of formaldehyde.

Test for Boric Acid or Borax.—To a few drops of the milk add a couple of drops of hydrochloric acid and a few drops of an alcoholic solution of turmeric in a white dish, and heat for a few minutes; the appearance of a red colour, changing to blue on cooling and adding ammonia, indicates the presence of boric acid or borax.

Evaporated Milk.—Under this heading are to be considered two kinds of milk, viz.:—

(*a*) *Dried or desiccated milk*, in which the milk is dried to a

powder. All such milks, which may keep indefinitely, have a practically uniform composition, viz.: Protein, about 25 per cent.; fat, 25 per cent.; sugar, 37 per cent.; salts, 7 per cent.; water, 6 per cent. In other words, the solids are present to the extent of about eight times the strength of the original milk, and hence, when used for infant feeding, must be diluted in the proportion of one drachm to the ounce in order to produce a food of the usual composition of milk.

Bacteriology of Dried Milk.—In the process of its manufacture the heat employed is enough to kill all the pathogenic organisms but not the spores. Moreover, as the powdered milk is not handled under aseptic precautions, fresh bacteria are added in subsequent manipulations. As mentioned above, however, the absence of water keeps the spores and the other organisms in check. If, however, moisture succeeds in penetrating the tin, moulds may begin to grow.

(*b*) *Condensed Milk.*—There are two varieties of this kind: (*a*) *Sweetened*, and (*β*) *unsweetened.*

- (*a*) *Sweetened condensed milk* is made in two forms recognised by the Public Health (Condensed Milk) Regulations of 1923:—
  - (i) Condensed full cream milk sweetened. This must contain at least 9 per cent. milk fat and 31 per cent. of all milk solids, including fat.
  - (ii) Condensed skimmed milk sweetened. This must contain 26 per cent. solids.
- (*β*) *Unsweetened condensed milk* is also manufactured in two recognised forms:—
  - (i) Condensed full cream milk unsweetened. This must contain at least 9 per cent. milk fat and 31 per cent. of all milk solids, including fat.
  - (ii) Condensed skimmed milk unsweetened, which must contain 20 per cent. of milk solids.

During the process of evaporation about $2\frac{1}{2}$ to $2\frac{3}{4}$ parts of milk are reduced to one part, and therefore when prepared for feeding purposes must be diluted accordingly.

The following **classification of infant foods** is modified from Hutchison.

Class I (which is the same as Group I in Table CXXXIV) consists of foods prepared from cow's milk with various additions

or alterations. They are prepared for use by the addition of water only. They are mostly poor in fat and too rich in carbohydrate.

Class II (same as Groups II and III in Table) consists of farinaceous foods prepared from cereals—usually wheat—of which the starch has been partly or wholly transformed into soluble substances. These are prepared for use by addition of milk and water. They may be subdivided into two groups:—

(*a*) Those in which the starch has been transformed before reaching the consumer, e.g., Mellin's Food, i.e., Group II in Table.

(*b*) Those in which the starch is transformed in the process of preparation for use, e.g., Benger's, &c., i.e., Group III in Table.

These foods are poor in fat, protein and mineral matter, and hence must be prepared for use by addition of some milk.

Group II is preferable to Group III, because in the latter the amount of starch that is converted into sugar is not constant, depending as it does upon the will of the person who mixes the food.

Class III (same as Group IV in Table) consists of farinaceous foods in which the starch has not been predigested.

Value of Bacterial Count.—It must be remembered that, though milk produced under ideal conditions should not contain more than 30,000 bacteria per cubic centimetre, yet a dairy bacterial count alone of any sample of milk means practically nothing, unless the milk is known to have been otherwise produced and distributed in the required manner. Thus a clean but unrefrigerated milk will in a very few hours show a much higher bacterial content than a dirty but properly cooled milk. For the same reason a milk delivered a long distance away from its source contains more bacteria than one delivered locally. In the hot summer period, also, the bacterial count is higher than during the cold winter months.

Vital Principles.—In addition to the physical, chemical, physiological and biological differences, each of which can to some extent be so adjusted as to make the two kinds of milk approximately alike, there are still certain vital differences between the two milks, the nature of which cannot be clearly defined but which nevertheless make each kind of milk specific for the young animal for which it has been intended by nature.

TABLE CXXXVI.—SHOWING THE COMPOSITION OF INFANT FOODS (AFTER HUTCHISON).

Food	Water	Protein	Fat	Carbo-hydrate	Mineral matter	Remarks
Dried human milk	—	12·2	26·4	52·4	2·1	Standard to which artificial substitutes should conform
Group I.						
Cow and Gate	4·3	25·2	26·4	37·9	6·0	Dried milk without addition of sugar
Allenburys No. 1 for babies under 3 months	5·7	9·7	18·5	62·3	3·75	Desiccated cow's milk from which excess of casein has been removed and to which soluble vegetable protein, lactose and cream have been added. No starch present
Allenburys No. 2 for babies 3 to 6 months	3·9	9·2	17·6	66·8	3·50	Similar to above, but contains also some malted flour. No starch
Horlick's malted milk	3·7	13·8	9·0	70·8	2·70	Similar to Allenburys No. 2
Almata	7·0	11·4	26·0	52·9	2·60	Composed of eggs, butter fat, dextri-maltose and fruit juice
Glaxo (full cream)	3·5	22·2	27·4	41·0	5·9	Dried milk together with lactose and cream
Group II.						
Mellin's food	6·3	7·9	Trace	82·0	3·8	A completely malted food
Group III.						
Savory and Moore's	4·5	10·3	1·4	83·2	0·6	Composed of wheat flour with addition of malt. When prepared according to directions, most of the starch is converted into soluble dextrin
Benger's food	8·3	10·2	1·2	79·5	0·80	A mixture of wheat, flour and pancreated extract, so that when prepared the starch and protein become partly digested
Group IV.						
Ridge's food	7·9	9·2	1·0	81·2	0·70	Contains most of carbohydrate in form of starch
Robinson's patent barley	10·1	5·1	0·9	82·0	1·90	Ground pearl barley, poor in every element except starch and mineral matter

For not only are the immune substances present in the two kinds of milk different, but each milk when injected into an animal will create a specific antibody in the serum of that animal which will precipitate that milk and no other milk.

The History of the Development of Artificial Feeding.—Amongst the ancients artificial feeding of infants with milk other than the mother's or that of a wet-nurse was practised very little. That it was used, however, is evidenced from the fact that not only have pictures of feeding-bottles been found engraved on stone, but the actual bottles themselves have been found in ancient Roman coffins of infants whom, as Pritchard remarks, "this method of feeding had done to death" (see figs. 14 and 14A, p. 40). Evidence of feeding-bottles with artificial nipples is found in the work of Soranus (second century A.D.). "One must take care," says Soranus (Chapter XXXIX), "not to give the baby any milk between feeds, for when the food (pap, &c.) floats on the top of the milk, it is distributed with difficulty throughout the body. Yet, if the baby is very thirsty, one may give it water or diluted wine from an *artificial nipple*."[1]

The nature of the artificial food given was butter, honey, milk, pap, &c. (see Chapter II, p. 38).

Metlinger, in 1473, mentions pap and milk as supplementary to breast feeding. He also advises cool boiled water or a little diluted wine. If weaning must take place prematurely (i.e., before two years), a mixture of three parts of boiled goat's milk and one part of boiled water should be given out of a cornucopia or a little jug.

After the fifteenth century artificial feeding became more common. It is said that out of 132 foundlings fed on diluted milk, gruels and broths, with sugar water and apple juice, only five lived to the age of 2 years. Cleanliness of the bottle was a recognised necessity in 1802.

Till the Middle Ages the idea was entertained (Xenophon, Aristotle, Theophrastus, Plato, Virgil, &c.) that the moral characters of an infant were determined by the nature of the milk it was taking, an idea still held by a few cranks at the present time and expressed by the German proverb, *Mann ist was er isst* (Man is what he eats). Romulus, for instance, was cruel because he was nursed by a she-wolf. Favorinus, of the second century A.D., affirmed that lambs nourished with the milk of goats have coarse wool, whilst kids suckled by sheep have woolly hair. Wherefore, it behoved not only that no other than human milk be given to a baby but that the mother

[1] Si forte siticulosus sit post cibum infans, aqua vel vinum aquosum illi praebendum est ope papillarum factitiarum.

and not a wet-nurse should suckle her infant. Thomas Phaer, of the sixteenth century, the father of English pædiatrics, writes in the same strain.

The fact, however, that notwithstanding our milk-drinking and non-vegetarian habits we still retain a vestige of human moral characteristics should suffice to convince most people of the absurdity of such a contention.

The Protein Period (Biedert, Meigs and Morgan Rotch).—The foundation of the scientific feeding of infants, artificially, was laid in 1869,[1] when Philip Biedert studied the chemical and physiological differences between human and cow's milk. Since Biedert's time each of the food elements in milk has in turn been indicted as the chief culprit in the causation of gastro-intestinal trouble in infancy. Biedert's studies revealed the fact that cow's caseinogen gave tough curds, on coagulation with rennin, and accused it of causing digestive disturbances in artificially-fed infants. He fortified his position by the observation that the stools of such babies contained hard curds, which he believed consisted of undigested casein curds formed in the stomach. On the basis of this theory he attempted to reduce the caseinogen, by dilution, to the percentage in which it is present in human milk. He introduced a cream, milk and sugar mixture fulfilling these conditions, especially with respect to the percentage of caseinogen. He called this mixture "*Rahmgemenge*" (i.e., cream mixture), and a condensed product of this is still on the market under the name of Ramogen.

This idea of reducing the caseinogen to the percentage present in human milk was elaborated, in 1885, by Arthur V. Meigs, of Philadelphia, who introduced "Meigs' Mixture," consisting of 1 oz. of milk, 2 oz. of cream (14 to 16 per cent.), 2 oz. of lime water and 3 oz. of milk sugar solution ($17\frac{3}{4}$ drm. of milk sugar to one pint of water). This has the following composition: Protein, 1·2 per cent.; fat, 3·5 per cent.; sugar, 6·7 per cent.; and salts, 0·25 per cent.—a composition practically identical with that of human milk.

Percentage Feeding.—Thomas Morgan Rotch, of Philadelphia, in 1889, went a step farther still. He introduced the idea of *percentage feeding*, which aimed at prescribing milk mixtures which contained the various ingredients, caseinogen, lactalbumin, fat and lactose, to suit the requirements of each individual infant. This idea was based on the supposition that the symptoms and signs of excess or deficiency of any of these components were accurately known, which, however, is far from being the case.

[1] *Virchows Arch. f. Pathol. Anat.*, lx, 1874, p. 352.

The method of percentage feeding was introduced into this country by Ralph Vincent, in 1903, when be began to feed all the patients at the, then newly-founded Infants Hospital, on this plan. Each individual infant was fed on a special formula, of which the following is a type :—

℞	Fat	1·50 per cent.
	Lactose	5·50 ,,
	Lactalbumin ..	0·50 ,,
	Caseinogen	0·25 ,,
	Alkalinity	5·00 ,,

Ten feeds of 4 oz. each.

The fat was supplied by cream, the lactose by a 20 per cent. lactose solution in water, the caseinogen by fat-free milk, the lactalbumin by whey, and the alkalinity by lime water. A prescription like the above would be prepared from these substances in the following proportions :—

℞	Cream (32 per cent.)	56 c.c.
	Fat-free milk	71 ,,
	Whey	556 ,,
	Lactose (20 per cent. solution) ..	151 ,,
	Lime water	60 ,,
	Water	325 ,,
		1,219 c.c.

Each feed would consist of 122 c.c., i.e., about 4 oz. However useful such a method is in a special hospital—and the author can speak from several years' experience of the considerable value of this method—it is one which is obviously difficult to carry out in private practice and is therefore very rarely used now.

The Fat Period (Czerny and Keller).—After a reign of many years the throne of caseinogen as the monarch in the domain of infant feeding began to totter. Clinical observations, as well as metabolic experiments, revealed the fact that not only was cow's caseinogen not difficult to digest, but that the curds that were sometimes found in the stools were small, soft and composed of fat, and not large, hard and consisting of casein, as described by Biedert. Czerny and Keller (1906-09) ultimately deposed caseinogen from its prominent position and enthroned cow's milk fat in its place as the principal factor in nutritional disturbances of infancy.

"There is no single symptom," say Czerny and Keller, "which can show us injury to the infant through protein. . . . It is possible that with abnormal flora in the intestine putrefaction

of casein *might* occur. It is, however, as yet unknown whether or not this actually happens."

The harmfulness of fat was stressed by George Armstrong (1755-1830), who deprecated the idea of cramming infants with fat "from a mistaken notion that it must be light of digestion, because it weighs light upon the scale; whereas, on the contrary, to most stomachs there are few things so hard to be digested as fat" (Rubräh). This however applied to infants with pyloric stenosis.

Homogenisation.—Those who attribute the indigestibility of cow's milk to the large size of its fat globules, advocate the use of milk in which the size of the globules is reduced by a pressure of 3,000 to 5,000 lb. to the square inch. This process is called *homogenisation* and has been extensively used in France. In addition, Ladd[1] adds 1·5 per cent. to 3·5 per cent. of olive oil to skimmed milk mixtures, before homogenisation, in order to render the fat still more similar to that of human milk. The value of both homogenised milk as well as of homogenised olive oil mixtures is still *sub judice*.

The Bacteriological Period (T. Escherich).—In the meantime the work of Escherich[2] (1885 onwards) and others on the intestinal bacteria focused attention to bacterial causes of diseases of infancy, especially summer diarrhœa. In cases of carbohydrate fermentation (acid and frothy stools and prevalence of lactic acid organisms in them), carbohydrate is to be withdrawn from the diet and protein substituted to change the type of the intestinal flora, whilst in cases of protein decomposition (offensive alkaline stools, with excess of proteolytic organisms in them), protein should be replaced by carbohydrate. This is probably the most important principle from the point of view of infant feeding.

Lactose and Salt Period (H. Finkelstein and Meyer, 1906 onwards).—In 1906 Finkelstein[3] absolved both protein and fat of all blame, and put forward the theory that sugar was the guilty party, on the ground that sugar and sodium salts when given to infants with intestinal trouble produce in them a rise of temperature.

The "whey exchange" experiments of L. F. Meyer and others (see p. 340) suggest that it is the whey salts of cow's milk which are responsible for the infant's digestive disturbances. They are believed to depress the antibacterial function of the cells of the small intestine, thus allowing excessive bacterial growth with consequent fermentation of sugar. The acid thus produced

[1] *Arch. Ped.*, xxxiii, 1916.

[2] "Die Darmbakterien des Säugling und ihr Beziehung zur Physiologie der Verdauung" (1886).

[3] "Ueber alimentare Intoxikation," *Jahrb. f. Kinderheilk.*, 65, 1907.

injures the intestinal mucosa, allowing the salts and unaltered lactose to pass through into the general circulation, where they have a toxic action. As stated on p. 344, however, Lichtenstein and Lindberg were unable to confirm Meyer's results, thus removing one of the corner-stones of Finkelstein's theory.

Protein Milk (*Eiweissmilch*).—Finkelstein and Meyer, believing as they do that it is the salts and sugar in the cow's milk which are responsible for intestinal disturbances of infants, have introduced *Eiweissmilch* to combat such disturbances. This food consists of a diminished quantity of lactose and of sugar, and an increased quantity of casein, together with varying quantities of fat. This food is prepared as follows:—

(1) Heat 1 quart of whole milk to 100° F.

(2) Add 4 teaspoonfuls of essence of pepsin or of rennet and stir.

(3) Allow the mixture to stand at 100° F. for about twenty to thirty minutes until the curd is formed.

(4) Strain off the whey (which is thrown away) through a linen cloth.

(5) Remove the curd from the linen cloth and press it through a rather fine sieve two or three times by means of a wooden spoon.

(6) Add 1 pint of water to the curd during this process.

(7) Add 1 pint of buttermilk (see p. 445) to this mixture.

The composition of this food is: Fát, 2·5 per cent.; lactose, 1·5 per cent.; protein, 3·0 per cent.; salts, 0·5 per cent.; and its calorific value is 12 calories per ounce.

The food is obviously of very low caloric value and can therefore be used temporarily only. Also it is very difficult to prepare it in the private home, and various manufacturers have prepared powdered protein milk. Mead, Johnson and Co.'s powder, when mixed with the proper amount of water, gives a food closely resembling the original formula in composition, and although it is hardly rational to use it in all forms of diarrhœa, as Finkelstein and Meyer advocate, it is very useful in cases of acid fermentation (when the stools are acid to litmus), as is also the substitution of maltose dextrin mixtures for lactose. Further, owing to its large content of lactic acid bacilli (from the buttermilk), it is probably of some use in checking the activity of proteolytic organisms and may, therefore, be of some use also in cases of intestinal intoxication.

Anhydræmia.

Still more recently the blame for nutritional disturbances of infancy has been laid on the **deficiency of water** in its food, resulting in anhydræmia.

When the water intake is diminished the blood becomes concentrated, as shown by a diminution in its volume, a rise in its specific gravity, corpuscular volume, and salt content, as well as by a rise in its electrical conductivity and osmotic pressure due to increased viscosity. Now Starling has shown that when the colloidal osmotic pressure of the blood exceeds the arterial blood-pressure in the renal glomeruli, suppression of urine occurs resulting in uræmia and acidosis. Hence

insufficient water intake may give rise to symptoms of acidosis and uræmia.

During hot weather the loss of water from the lungs and skin is greatly increased. Indeed, it has been shown that an increase of external temperature from 20° to 36° C. may increase water evaporation from the body by 600 per cent.[1] Hence high atmospheric temperature may give rise to anhydræmia unless a sufficiency of water is given to counterbalance the loss.

Atmospheric Temperature and Summer Diarrhœa.—Experiment has shown that prolonged exposure of young animals to high temperature produces diarrhœa and vomiting, and a great fall in the amount of gastric secretion, including free HCl, total acidity as well as pepsin and rennin. Hence it is believed by some that summer diarrhœa is due not so much to the influence of temperature upon the milk as to the influence of the heat upon the infant's own body. If this is true it follows that during the hot season all babies, whether breast- or bottle-fed, should receive less milk, because the gastric secretion is not sufficiently copious to digest the normal quantity, and the diminished acidity of the stomach permits an overgrowth of bacteria in an abnormal part of the bowel, such as the small intestine. It also follows that during the summer months the infant should take plenty of water and should be fed on acid milk or protein milk.

McKim Marriott[2] has demonstrated by G. N. Stewart's calorimetric method that whilst in normal infants the volume flow of blood in the arms (i.e., the number of cubic centimetres of blood per 100 grm. of body weight per minute) is 15 to 22 c.c., that in the arms of infants suffering from dehydration is only 2 to 3 c.c. per minute. If the volume flow in the abdominal vessels is diminished in the same proportion, it is obvious that the gastro-intestinal tract could not properly cope with the digestion of a normal amount of food. Marriott also found by the refractometric method[3] that the protein content of the serum, which in normal infants is 6 per cent. at 6 months and 7 per cent. at 1 year, is, in the case of dehydrated infants, about 50 per cent higher than normal.

It will be seen that anhydræmia is in some respects analogous to loss of blood, inasmuch as there is loss of blood volume in each case. The intoxication due to anhydræmia is, however, different from the shock caused by hæmorrhage because, whilst in the latter the blood volume is diminished on account of loss of all the blood constituents, in anhydræmia the diminution in volume is entirely due to loss of water. Hence, whilst in shock the protein concentration is low, and therefore also the blood-pressure is low, in the case of anhydræmia both the protein concentration and the blood-pressure are high.

It will thus be seen that since the beginning of the scientific study of infant feeding, some fifty years ago, every one of

[1] Langstein-Meyer, "Säuglingsernährung und Säuglingsstiffwechsel," Wiesbaden, 1914, p. 41.

[2] *Amer. Journ. Dis. Children*, 20, 1920.

[3] See W. M. Feldman, "Child Physiology," p. 423.

the constituents of milk has, in turn, been put upon its trial as the principal offender and been subsequently found not guilty. Indeed, those who are inclined to be cynical might, with some reason, quote this as an illustration of the saying of that wisest of men and gloomiest of pessimists: "And I gave my heart to know wisdom and to know madness and folly; I perceived that this also is vexatious of spirit. For in much wisdom is much grief; and he that increaseth knowledge increaseth sorrow" (Eccl. i. 17, 18).

The question that the thoughtful student will ask himself is: How can the various conflicting statements be reconciled, assuming the evidence on each side to be true and unbiased? How could Biedert, for instance, declare that the fæcal curds in babies fed on cow's milk were large and hard and consisted of casein, while equally trustworthy observers a few years later found in babies similarly fed that the curds were small and soft and were composed of fat? The answer has been given by Y. Ibrahim[1] and Joseph Bannerman[2] independently, who have shown that the curds in the stools of babies fed on *raw* milk have all the characters described by Biedert and are derived from undigested casein (as shown by analysis as well as immunological tests, and confirmed by H. Hess, who proved that milk introduced direct into the duodenum by means of a duodenal catheter, so that coagulation is avoided, never gives rise to such fæcal curds); whilst if the milk given to the baby has been previously *boiled* there are either no curds at all in the stools, or, if they are present, are small and soft and are composed of fat. It is probable, therefore, that Biedert's observations were made on infants fed on raw milk, whilst those of later observers, like Czerny and Keller, were fed on boiled milk. As regards Finkelstein's views, it may be remarked that the evidence in their favour has always been very scanty, since not only is bovine lactose identical in composition and other characters with the human variety, but cow's milk contains less of it than does human. Indeed, in 1910, Finkelstein himself abandoned the view that lactose was harmful (Morse and Talbot).

"The subject of infant metabolism and nutrition," says Garrison rightly,[3] "has been a debatable land made up of the

[1] *Monatschr. f. Kinderheilk.*, x, 1911.
[2] *Am. Journ. Dis. Childr.*, 1, 1911.
[3] Abt's "Pediatrics," vol. i, p. 132.

shifting sands of theory. The history of metabolism itself may be likened to a succession of dissolving views. The assemblage of different theories has all the effects of Schopenhauer's concert of Russian horns, each player producing (in his place) a single isolated note." The only facts which remain unassailed are: (1) The fact established by Rubner and Heubner (1898-99) that the daily nutritional requirements of an infant are to be represented by a certain number of calories per unit body weight; (2) the fact that each infant requires a minimum amount of protein.

Whole Milk Feeding.—Disheartened by the fairly rapid kaleidoscopic changes in the views regarding the blame to be attached to different milk constituents, certain authorities have revolted against the idea of cow's milk dilution, and are advocating the use of "whole milk" for babies.

Prof. Leonard Findlay, of Glasgow, is an enthusiastic exponent of this doctrine in this country, and Budin, Variot, Rothschild, and others, used it extensively in France. The following are arguments given against dilution: (1) Dilution, whilst it will reduce the total protein to the same proportion as found in human milk, will also reduce the valuable lactalbumin which contains a high percentage of the essential amino-acid tryptophane; (2) the caseinogen, even when undiluted, has no bad effect upon the baby, as proved by the "whey-exchange" experiments (p. 340), as well as by the fact that the fæcal curds in cases of digestive disturbance do not consist of casein but of fat; (3) further, dilution reduces also the percentage of these substances that are already present in smaller quantities in cow's milk; (4) moreover, clinical experience, according to Budin and Findlay, shows that normal infants, as a rule, digest and thrive well on undiluted milk.

It is to be remarked, however, that whilst very strong babies can digest anything, it does not prove that all babies can take whole milk with impunity. Indeed, from what we have stated regarding the quantitative and qualitative chemical differences between cow's and human milk, it would be very surprising if cow's milk could without modification take the place of the human variety. The matter has been tested by Marfan[1] and he finds as follows:—

(1) Infants with digestive troubles (at the Hôpital des

[1] "Traité de l'allaitement," Third Edition, 1920, Paris, p. 557.

Enfants Malades, in Paris) get their symptoms of vomiting and diarrhœa frequently aggravated, but never diminished.

(2) Healthy babies, after the sixth month, can take it quite safely.

(3) Of the healthy babies under six months :—

(*a*) A minority take it well and thrive on it.

(*b*) A large majority show symptoms of protein excess, viz., their weight becomes stationary, they begin to look pale, and their stools, of which there are several a day, are pasty, homogeneous, pale yellow, offensive in odour, and have an alkaline reaction. Although such symptoms may occur with diluted milk, they are much less frequently met with.

(*c*) Another group suffer from vomiting and diarrhœa.

(4) Babies under three months cannot be fed at all on "whole milk." This is also to some extent admitted by Findlay.

In addition to the theoretical arguments against the use of whole milk, based upon the quantitative composition of cow's milk, two others deserve mention, viz., water deficiency and high buffer value.

Water Deficiency.—We have seen that the protein requirement of a normal baby is 1·5 grm. per kilogramme body weight. This amount would be furnished by about 3 oz. of human milk or $1\frac{1}{2}$ oz. of cow's milk per pound weight of the baby. But we have also seen that the water requirement of the baby is 3 oz. per pound weight, so that a baby fed on whole milk only gets half the quantity of water it needs unless it is given more milk than it is able to digest. This argument also applies to whole milk to which citrate of soda has been added to render the caseinogen more digestible.

High Buffer Value.—By the buffer value of a solution is meant its capacity to unite with an acid or an alkali without undergoing a corresponding change in its chemical reaction. Thus, a solution containing bicarbonate of soda, $NaHCO_3$, when uniting with HCl, will go through the following reaction,

$$NaHCO_3 + HCl = NaCl + CO_2 + H_2O,$$

and as CO_2 when dissolved in water is a very *weak* acid, therefore the reaction of the solution will remain practically unchanged. Phosphates and amino-acids behave in the same way.

It has been found that the buffer value of human is less than that of cow's milk, because it contains less phosphates, so that the

same amount of HCl added to equal quantities of the two milks will increase the acidity of the human to a greater extent than that of the bovine variety. Now the gastric acidity of a normal breast-fed infant after a feed is expressed in H-ion concentration as $C_H = 1 \times 10^{-5}$, which, according to Hahn, is the optimum concentration for the action of rennin and for the inhibition of bacterial growth. But if such an infant were to take cow's milk whose buffer value is greater, the secretion of the same amount of gastric juice is insufficient to render the stomach content acid to the same degree, and in order to produce the optimum acidity, a secretion of at least three times as much HCl is required as in the case of human milk. Such excessive secretion is beyond the functional capacity of the baby's stomach, and hence not only is bacterial growth not inhibited, but the rennin action occurs to a slight degree only, and the amounts of bile and pancreatic juice whose secretion depends upon the amount of gastric acidity becomes greatly diminished. Hence, whole cow's milk is theoretically contra-indicated for babies. Indeed, feeble babies tolerate best those foods which either have a low buffer value, or whose buffer action has already been partly neutralised by acid. Breast milk, lactic acid milk and protein milk are examples.

Modification of Milk.—Whilst the contention of the "whole milk" advocates is certainly true, that simple dilution does not solve the problem, modification in such a way as to bring the various constituents as nearly as possible to the proportions in which they are present in human milk, though it cannot convert cow's into human milk, at least attempts to accomplish this as far as it is possible to do so.

The Use of Cow's Milk for Infants.—The question of the proper use of cow's milk in the artificial feeding of infants is, as we have seen, a very intricate one. It is a vast battlefield in which every inch of territory has been and is being keenly disputed. From what has been said in the foregoing pages, it will be realised that the number of differences between human and cow's milk is very large, for not only are the two kinds of milk different in quantitative and qualitative composition, but they are totally different biologically, immunologically and bacteriologically. Yet the significance of each or all of these differences has, as we have seen, been interpreted differently by different authorities. The various views will be summarised here in the briefest possible manner, and an endeavour will be made to give a verdict in the direction indicated by the balance of the evidence.

(1) **Breast versus Cow's Milk Feeding.**—Although it would appear obvious that breast-feeding must be incomparably superior to bottle-feeding, yet there are some who believe that the greater mortality of bottle-fed babies, as compared with those nursed by their mothers, is merely apparent and due to the fact that the breast-fed babies are hereditarily of a superior stock. The question has been discussed in Chapter V, p. 153, where the fallacy of such a contention has been pointed out. **There can be no doubt that breast milk is the best that can be given to a baby, and that cow's milk may be given only when human milk is unavailable.**

Herd Milk versus one Cow's Milk.—Inasmuch as it is easier to keep one cow clean and free from tuberculosis than a whole herd, there seems an obvious advantage in using one cow's milk for infant feeding in preference to herd milk. On the other hand, since the composition of milk is so variable, and the danger of failing to detect tuberculosis in a cow is very considerable, herd milk, representing as it does a mixture of milks from numerous cows, must necessarily give a milk of average composition and also ensures great dilution of bacterial concentration, including that of the tubercle bacilli, thus rendering it much safer for consumption by infants. Experiments show that large doses of virulent tubercle bacilli are required to bring about infection through the intestine. Further, if the cow from which the milk is taken happens to be ill in any way, the milk might disturb the baby, but if the milk is mixed with that of other cows, the toxin becomes diluted and is not felt by the baby.

Should Milk be Heated?—Whilst nobody disputes the advisability of procuring a clean and "pathogenic germ-free" milk, there is considerable difference of opinion regarding boiling or pasteurisation of milk. The advocates of raw milk contend :—

(1) That breast milk is taken by the baby raw. Against this, however, are to be put the facts that : (i) Breast milk is nearly sterile and at any rate free from pathogenic organisms, whilst cow's milk may teem with germs, including the tubercle bacilli, and boiling or pasteurising kills these germs ; (ii) boiling alters the character of the casein curds and makes it easier for the infant to digest.

(2) Raw milk contains antitoxins, antibodies, and vitamins, some of which are destroyed by heat. But it must be remembered that : (i) Antibodies in bovine milk can be of use only to the calf and not to the human infant ; (ii) vitamin A is not

destroyed by heating, whilst vitamin C, though destructible by prolonged boiling, can be supplied by means of grape juice, &c.

(3) Boiling kills the milk-souring germs, viz., the *Streptococcus lacticus*, *Bacillus acidi lactici*, &c., which have an inhibitory effect upon the growth of the true putrefactive organisms, many of which are spore-forming and survive prolonged boiling. This is a very strong argument against boiling milk, but, on the other hand, milk so treated can only be harmful if allowed to stand any considerable time before using, since the spores might develop in the meantime. If, however, milk is used immediately, such a danger does not arise unless, as the result of intestinal stasis, the swallowed spores stay long enough inside the intestine to develop. Hence milk should be boiled at home and not at its source.

As a clinical example of the antagonism between the milk-souring and putrefactive organisms, the one recorded by Lorenz, in 1920, is very instructive. Lorenz described an outbreak of Y dysentery in a Hamburg Orphan Asylum, which he believed was traceable to milk, which after boiling was infected with *Bacillus dysenteriæ* Y in the orphanage kitchen. He brings experimental evidence to show that although raw milk is a very bad culture medium for the growth of the dysentery bacillus, the destruction by sterilisation of the bacteria in the original milk renders the latter a good medium for the growth of the dysentery bacilli.

Experiments with Animals.—Ralph Vincent, who was the protagonist of raw milk for babies in this country, fed kittens on pure milk which had been raised to a temperature of 200° F. All these kittens died. From this he drew the conclusion that boiling of milk is contra-indicated in infant feeding.

The criticisms that can be made on these experiments are: (1) In addition to heating the milk to 200° F., Vincent also incubated it for twenty-four hours at 85° F. before administration, a thing which is never done in ordinary nursery routine; (2) no control experiments were made with kittens fed on raw milk, and as cat's milk is, on account of the animal's rate of growth, of totally different composition from that of the cow (the kitten doubles its birth weight in seven days, and cat's milk contains 9·5 per cent. of protein, as against the 3·5 per cent. of protein in cow's milk), it would *a priori* be expected that young kittens would not thrive on cow's milk whether boiled or raw.

Many experiments have been carried out on different animals (e.g., guinea-pigs, rats and mice, rabbits, dogs, pigs and goats) by different observers, which on the whole did not demonstrate any appreciable difference in nutritional value between raw and boiled

cow's milk, but all such experiments are open to the criticism that the cow's milk with which the experiments have been carried out is totally different in composition from that of the animal's own species.

Observations in Infants.—In order to draw any very reliable conclusion with regard to the relative merits of the two kinds of milk, observations under experimental comparable conditions must be made on babies. Such experiments as have been carried out (e.g., Finkelstein, Park and Holt, and others) do not on the whole show any difference between raw and boiled milk from the nutritional standpoint, although the figures of Park and Holt show that out of 41 infants fed on pasteurised milk 10 developed diarrhœa (i.e., 25 per cent.), whilst out of 51 fed on raw milk, 33 had diarrhœa (i.e., 65 per cent.). The most extensive observations on a comparable basis of the comparative values of boiled and raw milk was carried out by Medin, in the Orphan Asylum at Stockholm.[1] He fed half the number of nurslings on raw milk and the other half on the same milk which had been boiled. During a period of three years no difference could be recognised between the two groups.

Summary.

If we summarise what we have said with regard to the question of heating milk, we may say:—

(1) Under no circumstances should the boiling of milk be made a substitute for clean milk.

(2) When clean milk is reasonably assured it is safer to boil it, because:—

- (i) Boiling or pasteurisation kills the tubercle bacilli, if present.
- (ii) Boiling kills the germs of other infective diseases.
- (iii) It is true that boiling kills all the fermentative organisms and not all the putrefactive ones, but the risk of causing intestinal putrefactive trouble is nil if the milk is given soon after boiling. The risk may be still more minimised by giving the baby some culture of *Streptococcus lacticus* with its cooked milk. (See under Lactic Acid Milk, p. 445.)
- (iv) There is no appreciable nutritive difference between raw and boiled milk.

[1] Quoted by Finkelstein, "Lehrbuch der Säuglingskrankheiten," Berlin, 1924, p. 36.

(v) Vitamin A is not destroyed, and possible destruction of Vitamin C can be made good by means of fruit juice.

(vi) Boiled milk is more digestible than raw.

(3) After boiling the milk must be kept cool, and covered and handled with the greatest care to keep out extraneous organisms.

Formulæ for Diluting Cow's Milk to Make it of any desired Composition.

Supposing we wish to dilute cow's milk to make it equal in composition (as regards protein, fat and carbohydrate) to that of human milk, we proceed as follows :—

If we dilute 10 oz. of the milk with water up to 20 oz. we get a mixture whose composition is : Protein, 1·75 per cent. ; fat, 2 per cent. ; sugar, 2·5 per cent. The protein is in about the proper proportion, but there is a shortage of 2 per cent. of fat and 4·5 per cent. of sugar.

If we add 0·9 oz. of sugar to the 20 oz. we add of course the 4·5 per cent of sugar. Similarly, 0·4 oz. of fat added to the mixture adds 2 per cent. of fat. But centrifugalised cream contains 50 per cent. of fat, therefore 0·8 oz. of the cream will contain the 0·4 oz. of fat. Hence the mixture must be made up as follows :—

Cow's milk	10 oz.	
Cream (50 per cent. fat)	0·8 ,,	(= 6½ drm.)
Sugar	0·9 ,,	(= 7 ,,)
Water up to	20 ,,	

If the baby weighs, say, 10 lb., it requires 15 oz. of undiluted cow's milk (i.e., 1½ oz. per pound) to give it the necessary amount of protein in twenty-four hours. Therefore the amount of diluted milk necessary in a day is 30 oz. This is divided into six feeds of 5 oz. each.

Such a mixture corresponds approximately to human milk in quantitative but not in qualitative composition. The principal qualitative difference, from the digestive point of view, is that the caseinogen in the cow's milk is not physiologically adapted to the baby's stomach. This can be remedied in several ways, either by substituting 1 oz. of lime water for ordinary water in each pint of the mixture, or by boiling the milk, or by adding sodium citrate, about 1 gr. to the ounce of the mixture, or by using dried milk (see p. 406).

But even quantitatively the mixture is not the same as human milk, since, although it contains approximately the same

proportion of total protein, each of the two proteins is present in different proportions from those found in human milk. Moreover, the various salts are present in totally different percentages.

TABLE CXXXVII.

Protein percentage			The fat percentage				
			1·5	2·0	2·5	3·0	3·5
1·0 per cent.	Percentage composition	Protein	1·0	1·0	1·0	1·0	1·5
		Fat	1·5	2 0	2·5	3·0	3·0
		Sugar	5·0	5·0	5·0	5·0	5·0
	Ingredients	Milk	5 oz.	4½ oz.	4 oz.	3¾ oz.	3¼ oz.
		Cream	3 drm.	6 drm.	1 oz.	1¼ oz.	1¾ oz.
		Sugar	6½ drm.	6½ drm.	6½ drm.	6½ oz.	6½ oz.
	Caloric value per oz.		11·5	12·5	13·5	14·5	15·5
1·5 per cent.	Percentage composition	Protein	1·5	1·5	1·5	1·5	1·5
		Fat	1·5	2·0	2·5	3·0	3·5
		Sugar	5·0	5·0	5·0	5·0	5·0
	Ingredients	Milk	7½ oz.	7½ oz.	7 oz.	6½ oz.	6 oz.
		Cream	1 drm.	3½ drm.	6 drm.	1 oz.	1½ oz.
		Sugar	5½ drm.	5½ drm.	5½ drm.	5½ drm.	5½ drm.
	Caloric value per oz.		12·5	14	15	16	18
2·0 per cent.	Percentage composition	Protein	2·0	2·0	2·0	2·0	2·0
		Fat	1·5	2·0	2 5	3·0	3·5
		Sugar	5·0	5·0	5·0	5·0	5·0
	Ingredients	Milk	Impossible	10 oz.	10 oz.	9 oz.	8½ oz.
		Cream		1 drm.	3½ drm.	7 drm.	1¼ oz.
		Sugar		5 drm.	5 drm.	5 drm.	5 drm.
	Caloric value per oz.		—	15·5	16·5	17	18·5
2·5 per cent.	Percentage composition	Protein	2·5	2·5	2·5	2·5	2·5
		Fat	1·5	2·0	2·5	3·0	3·5
		Sugar	5·0	5·0	5·0	5·0	5·0
	Ingredients	Milk	Impossible	12½ oz.	12½ oz.	12 oz.	11½ oz.
		Cream		None	1½ drm.	5 drm.	1 oz.
		Sugar		½ oz.	½ oz.	½ oz.	½ oz.
	Caloric value per oz.		—	16·5	17·5	18·5	19·3

Milk "Humanisation."

It is true that: (1) By the use of whey mixtures (see p. 442) we can adjust the proportions of caseinogen and lactalbumin to those in human milk; (2) we can make the size of the fat globules the same as in human milk by homogenisation (see p. 430); (3) we can alter the composition of the fat to make it similar to that in human milk; (4) we can also make the composition of the salts the same as in human milk. But it is equally true that: (*a*) The specific serum reaction and other biological properties cannot be changed; and (*b*) not all these

various changes can be made at the same time. If they could, "the cow would certainly not recognise it as her own product, and the baby would never mistake it for human milk" (Morse). Indeed, no matter what we do to cow's milk we can never make it the same as the human variety, and, as John Burns once pithily put it, "The only way to humanise cow's milk is to pass it through the mother."

The preceding Table CXXXVII (from Pritchard) gives formulæ for preparing milk mixtures of definite percentage composition (the cream used contains 33 per cent. of fat).

Whey.—In cases where caseinogen cannot be digested, it is possible to feed the infants on caseinogen-free milk or whey, which is prepared as follows :—

(1) Add a teaspoonful of rennin to a pint of fresh milk.

(2) Warm the **milk** (with the rennin), in a clean jug surrounded by water in a saucepan, to a temperature of 100° F., and remove the saucepan from the fire.

(3) Stir the milk in the jug until the curds are well-formed, at the same time press them to the bottom of the jug with a spoon so as to remove any fat collected in the meshes of the clot. It takes about ten minutes for the milk to curdle.

(4) When the curds are formed into a solid mass at the bottom of the jug, place the saucepan again on the fire and boil the water round the jug for one minute. This raises the milk in the jug to 150° F. and destroys the rennin, thus preventing its further action upon the milk.

(5) Pour off the whey (i.e., the milk-like fluid which remains) and keep it in a cool place, preferably over ice.

Whey prepared from whole milk as above is called **fat whey**. It is also possible to prepare fatless whey by subjecting skimmed milk to the same process.

The composition of each variety of whey is as follows :—

	Casein	Lactalbumin	Fat	Lactose
Fat whey	0	0·5	3 5	5
Fatless whey ..	0	0 5 (about)	0·1	5

Use of Whey Mixtures.—By means of mixtures of whey with milk and water, or sugar water, it is possible to prepare a feed for a baby containing a desired percentage of caseinogen and lactalbumin. The simplest (though not the most accurate) method is to use fat whey.

Examples.—Supposing we wish to prescribe a food containing 1·0 per cent. caseinogen, 0·5 per cent. and 3·5 per cent. of fat, we proceed as follows : Cow's milk contains :—

Fat, 3·5 per cent.
Lactalbumin, 0·5 per cent.
Caseinogen, 3·0 per cent.

Take caseinogen first: Of the three components, milk, fat whey and water, milk is the only one that contains caseinogen, but since the percentage of this is 3, and we want to give only 1·0 per cent., therefore the amount of the milk must constitute one-third of the whole mixture.

Take the fat next. This is contained in both milk and fat whey in the same proportions, i.e., 3·5 per cent.

Let x = quantity of fat whey, then we have the following equation:—

$$3{\cdot}5\left(\frac{1+x}{3}\right) = 3{\cdot}5$$

i.e., $1 + x = 3$, whence $x = 2$.

Hence our prescription reads as follows:—

Fat whey	... ...	2 parts
Milk ...	... ...	1 part
Water...	... ...	nil

The following is **another example**: The food is to contain 0·4 per cent. of caseinogen, 0·3 per cent. of lactalbumin, and 2·0 per cent. of fat.

Since cow's milk contains about 3·0 per cent. of caseinogen, therefore it must constitute only one-seventh part of the mixture in order to give 0·4 per cent. of caseinogen. Hence, if x = quantity of fat whey, we have (for 2 per cent. of fat):—

$$3{\cdot}5\left(\frac{1+x}{7}\right) = 2, \text{ i.e., } 0{\cdot}5\,(1 + x) = 2,$$

or $1 + x = 4$, whence $x = 3$

Therefore we prescribe—

Fat whey	... ...	3 parts
Milk ...	... ...	1 part
Water...	... ...	3 parts

We can always calculate the **composition of any given whey** mixture as follows: Supposing we have a mixture of: Fat whey, 3 parts; milk, 2 parts; and water, 5 parts, the whole mixture consists of 10 parts. The caseinogen is present in the cow's milk only, i.e., in two-tenths of the mixture, therefore the percentage of caseinogen is $\frac{2}{10} \times 3 = 0{\cdot}6$ per cent. The fat is present in approximately the same proportions in the milk and

whey, which together constitute five-tenths of the mixture. Therefore the percentage is $\frac{5}{10} \times 3{\cdot}5 = 1{\cdot}75$ per cent. The lactalbumin is present in the whey and milk, i.e., in three-tenths of the mixture, therefore the percentage is $\frac{5}{10} \times 0{\cdot}5 = 0{\cdot}15$ per cent. The sugar, again, is present in the milk and whey in the same proportions, therefore its percentage is $\frac{5}{10} \times 5 = 2{\cdot}5$ per cent. Hence the mixture contains 0·6 per cent. caseinogen, 0·15 per cent. of lactose, 1·75 per cent. of fat and 2·5 per cent. of sugar. Its caloric value would, therefore, be $\left(\frac{0{\cdot}6 + 0{\cdot}15 + 2{\cdot}5}{100}\right) \times 116 + \frac{1{\cdot}75}{100} \times 263 = 8{\cdot}5$ calories per ounce, or twenty-nine calories per 100 cubic centimetres.

The following Table CXXXVIII gives formulæ for preparing whey mixtures containing various percentages of caseinogen and the other substances :—

TABLE CXXXVIII.

Percentage				Ingredients in parts			Calories per 100 c.c.
Caseinogen	Lact-albumin	Fat	Sugar	Fat whey	Milk	Sugar water (5 per cent.)	
0	0·5	3 5	5	1	0	0	53·5
0·3	0·3	2·1	5	5	1	4	41·3
0·4	0·3	2·0	5	3	1	3	40·8
0·5	0.3	2·3	5	3	1	2	73·9
0·6	0 4	2·8	5	3	1	1	49·2
0 7	0·4	2·6	5	2	1	1	47·8
0·8	0·5	3·5	5	3	1	0	56·7
0·9	0·45	3·2	5	6	3	1	54·2
1·0	0·33	2·3	5	1	1	1	48·0
1·0	0 33	3·5	5	2	1	0	56·8
1·5	0·5	3·5	5	1	1	0	59·5

The percentage of sugar can of course be varied by using different strengths of sugar solution, or by using ordinary water and adding the sugar subsequently, remembering that every 1½ drm. to the pint, or 10 grm. to the litre, raises the proportion of sugar by 1 per cent. (one level teaspoonful of cane sugar, and one and a half level teaspoonfuls of milk sugar, each weigh 1 drm.).

The **intervals between feeds** and the **amount of each feed** are the same as in the case of breast-feeding (pp. 386 and 387). For details about cleanliness see p. 157. The **teat** of the bottle must have a hole of suitable size—not too small to prevent the baby taking the milk without undue effort, nor too large to allow the baby to gobble up the milk too quickly. The right size is one which will allow the milk from the inverted half-filled bottle to drop out at the rate of a drop per second. Great care must be taken of its cleanliness.

Buttermilk and Lactic Acid Milk.

Buttermilk is sour milk from which the fat has been almost entirely removed. It is usually obtained as a by-product in the process of making butter. It has the following composition: Protein 3 per cent., fat 0·5 per cent., lactose 4·8 per cent., calorific value 10 calories per ounce. Its sour taste is due to lactic acid, and it also contains an abundance of the *Streptococcus lacticus*. Its low fat value renders it useful for babies who cannot digest fat. Its high acidity makes it valuable in feeble babies in whom the gastric secretion is not very active (see p. 432). Also its high lactic acid bacteria content renders it of particular value in cases of diarrhœa with offensive alkaline stools (see p. 408), i.e. in the opposite condition to that in which protein milk is used. This milk has been used in Holland for cases of infantile diarrhœa for several hundred years.

Lactic acid milk, which is used for the same conditions as buttermilk, is prepared from boiled skimmed milk which has been cooled to 70° F. by adding either tablets or liquid cultures of *Streptococcus lacticus* obtained from drug firms. The liquid culture is preferable because the tablet is sometimes inactive. The milk is then covered and kept at 70° F. for about twelve to fifteen hours. After that time 3 tablespoonfuls are removed and kept for the next day's preparation. The milk is now beaten with an egg-beater until it is smooth, but not frothy.

The Use of Milk of other Animals than the Cow in Infant Feeding.

Goat's milk is used extensively in Switzerland and other parts of the world where this animal predominates. In certain respects goat's milk has certain advantages over cow's: (1) The goat produces twice as much milk, in proportion to its body weight, than the cow; (2) the initial cost of a goat is small; (3) the

upkeep of a goat is small; (4) the goat is said to be immune to tuberculosis, although this is probably due to less insanitary herding than that of cows; (5) because a goat can be kept on the premises, the danger of contamination of its milk is very greatly reduced.

The disadvantages are: (1) From the point of view of digestibility and chemical composition, goat's milk offers no advantage over cow's milk (see Table CXXIX, p. 402), and according to some the casein clot is a firmer mass than that of cow's casein; (2) goats, especially in tropical countries, are the carriers and conveyors of Malta fever.

Asses' milk has been used in Spain and Italy and other countries. The advantage of this milk is that, as far as protein and carbohydrate are concerned, its composition is practically identical with cow's milk. They also very rarely suffer from tuberculosis. On the other hand, it is very poor in fat (see Table CXXIX, p. 402). Further, in proportion to their weight asses yield much smaller amounts of milk than do cows, and hence the milk is rather expensive. Indeed, in France, where the direct nursing from asses' udders was practised in the time of Parrot (see p. 49), it was given up on account of the expense, as it cost over 2,000 francs a year to feed one baby.

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CHAPTER XVII

THE FEEDING OF OLDER CHILDREN

"The feeding of children properly is of much greater importance to them than their clothing. We ought to take great care to be right in this material article and that nothing be given them but what is wholesome and good for them and in such quantity as the body calls for towards its support and growth; not a grain more. . . . If we follow Nature we cannot err. In the business of Nursing as well as Physick, Art is ever destructive if it does not exactly copy the original."

William Cadogan, 1769.

In Chapter XIII, p. 356, *et seq.*, we discussed fully the essentials of a diet for a growing child. We saw that it must fulfil certain qualitative and quantitative needs in order to be suitable for the child. The qualitative requisites are those with respect to digestibility, taste, ballast (or bulk), vitamin content, &c., whilst quantitatively it must supply enough protein to satisfy the needs of the body for wear and tear as well as growth, and it must also supply a sufficient number of calories per day, properly distributed between the protein fat and carbohydrates. In this chapter we shall deal with the problem of constructing dietary sheets for children of different ages on these lines.

Qualitative Considerations.

Digestibility.—Whilst some children can digest anything, the large majority of children can only deal with those articles of food which do not irritate the mucous membrane of the gastro-intestinal tract. The following articles ought to be eliminated from the menus of children under 5 or 6 years of age:—

(*a*) Foods which contain much insoluble cellulose, e.g., mushrooms, radishes, cucumber, celery, beetroot, nuts, plums, greengages, the skin of baked potatoes, currants, &c.

(*b*) Foods which contain hard resisting seeds, e.g., raisins, strawberries, gooseberries, raspberries, &c.

(*c*) Foods which require much effort in chewing, e.g., steaks, hard cheese, &c.

(*d*) All seasoned foods, such as salt beef, herrings, &c.

(*e*) Tea and coffee is to be given in great moderation and all alcoholic drinks are to be absolutely forbidden—except perhaps in cases of illness at a doctor's orders.

Ballast.—If a diet is too concentrated, it does not distend the bowels sufficiently to drive on the contents, with the consequence that constipation with all its attendant discomforts and dangers results. In herbivorous animals, whose diet consists of grassy food of very low calorific value, the amount of food they have to take to supply them with enough calories must be very large. In them the intestines are very huge in order to accommodate the bulky food material. If such animals are fed on less bulky food—such as corn which is of comparatively high calorific value—they become ill as the result of toxic absorption from the bowels, because their intestines lack the necessary ballast to expel any undigested contents. The human body, in the same way—but in much less degree—requires a certain amount of soft vegetable food of low calorific value and not of too irritable a character to distend the intestine sufficiently. Such foods are vegetable marrow, cauliflower, cabbage, turnips, potato, Brussels-sprouts, spinach, lettuce, &c. In addition to giving bulk they also contain a large proportion of mineral, especially iron, and of vitamins.

Taste.—Although children should not be forced to take those foods for which they exhibit a definite and decided dislike, it must be remembered that most tastes are acquired, and with proper training a young child will learn to like most of the articles of food offered to it. Very often a dislike for a certain food is acquired because of certain associations such a food has in its mind with certain unpleasant incidents. For example, if a particular food has been given to conceal the taste of an unpleasant medicine, an association between the food and the medicine may become established which it may become exceedingly difficult to break. In the matter of taste, therefore, suggestion plays a great part in the case of children, and the younger the child the more susceptible is its brain to the influence of suggestion. It is, however, very desirable to ensure that there shall be no monotony in the meals, and the menu ought to be frequently changed.

Vitamins.—These have been sufficiently discussed in Chapter XIII, but it is worth pointing out that summer milk, owing to the green food available to the cows, is richer in vitamin D than winter milk. Also in virtue of the smaller amount of natural ultra-violet rays, which are the best substitute for

vitamin D, in the winter, a diet richer in that vitamin is necessary in the winter months. Hence, while margarine may to a certain extent replace butter in the summer, it must not find a place in a child's dietary in the winter.

Quantitative Considerations.

(*a*) *Total Calories.*—We have seen that the human body, like an engine, requires fuel to keep it going, and in the same way as we calculate how much coal must be burnt to produce a certain amount of heat (e.g., to cook, or to drive an engine, &c.), so also is it necessary to calculate the total amount of food necessary to supply the needs of the human furnace with the requisite amount of heat.

(*b*) *Amounts of Protein, Fat, and Carbohydrates.*—These have been fully discussed in Chapter XIII, where it was shown that between the ages of 2 and 6 years the amounts required were 4 grm. each of protein and fat, and 12 grm. of carbohydrate per kilogramme body weight per day (see p. 366).

(*c*) *Amount of Water.*
(*d*) *Amount of Salts.*
} These have been considered in Chapter XIII.

Calculation of a Diet Sheet.—In order to prepare a dietary containing a mixture of several articles of food which would satisfy the child's requirements with regard to the proper quantities of protein, carbohydrate, fats, &c., and therefore also with respect to the number of calories, one proceeds as follows: (1) Decide about the amount of milk the child is to take during the day. This will account for some of the food ingredients as well as for the calories. (2) Make up a list of the additional food substances you are going to use (excluding those which are unsuitable for any reason), together with their quantitative chemical composition. (3) Ascertain the quantitative chemical composition of any of the dishes made up out of these food substances. This gives you at once the calorific value of these foods or dishes. (4) By the use of very easy arithmetic one can then work out the exact quantities of the different food substances required. (5) The matter becomes simpler still if the calculation is limited to one meal, instead of to the food requirements for the whole day.

The younger the child and the fewer the food substances that are chosen to supplement the milk, the simpler, of course, is the calculation.

It will be remembered that the calorific values are: Protein

and carbohydrate, 116 per oz. (or 4 per grm.), and fat, 263 per oz. (or 9 per grm.).

Diet for an Infant aged 9 to 12 months.—As a simple example we shall work out a dietary for an infant that has just been weaned. Such an infant obviously requires for the most part milk. We wish, however, to introduce some starchy food such as groats, barley, semolina, &c., and in addition we have to give it some solid food, such as rusk, to exercise its teeth, jaws, and chewing muscles. The caloric requirements of such an infant, assuming it to weigh about 18 lb., are $18 \times 45 = 810$ calories per day. This would be contained in approximately 2 pints of milk. Therefore, in order to introduce the other food substances we must give less than 2 pints of milk. Let us decide to give it $1\frac{1}{2}$ pints, and supplement the extra calories, &c., by means of semolina, butter, sugar and rusk.

TABLE CXXXIX.

Gruel	Protein	Fat	Carbo-hydrate	Calories
Milk, 6 oz. ..	0·21 oz.	0·21 oz.	0·30 oz.	114
Semolina, $\frac{1}{2}$ oz.	0·06 ,,	trace	0·40 ,,	54
Butter, $\frac{1}{4}$ oz. ..	—	0·25 oz.	—	66
Sugar, $\frac{1}{3}$ oz. ..	—	—	0·33 ,,	38
Total ..	0·27 oz.	0·46 oz.	1·03 oz.	272

If this is given as one feed, there will remain 24 oz. of milk for the other four feeds of 6 oz. each.

Milk, 24 oz. ..	0·84 oz.	0·84 oz.	1·20 oz.	456
Total for all feeds	1·11 oz.	1·30 oz.	2·23 oz.	728

But the infant requires 810 calories, therefore the remaining 82 calories must be made up from rusks.

Rusk, 1 oz. ..	0·10 oz.	0·01 oz.	0·59 oz.	83
Grand total ..	1·21 oz.	1·31 oz.	2·82 oz.	811

The diet for the day will therefore be arranged as follows:—

7 a.m.—6 oz. of milk and $\frac{1}{2}$ oz. of rusk.
10 a.m.—6 oz. of milk only, and no rusk.
1 p.m.—6 oz. of gruel.
4 p.m.—6 oz. of milk and $\frac{1}{2}$ oz. of rusk.
7 p.m.—6 oz. of milk only.

The usual number of feeds is five a day. We shall, to start with, give four feeds of pure milk, to which some dry rusk is added as a separate article (not soaked in the milk), and one feed we shall give of thin gruel or pap prepared from milk, semolina, butter and sugar. A gruel of suitable consistency (enough for one

feed) would be prepared as follows: Add ½ oz. of semolina, or cornflour, &c., to a little cold water and put the mixture into 6 oz. of milk and boil for twenty minutes. Then add a pinch of salt, ¼ oz. of butter and about ⅓ oz. of sugar. (The boiling renders the starch more easily digestible.) Such a gruel will have the composition shown in Table CXXXIX.

In addition, it is necessary to give an extra 6 to 8 oz. of water, since the total fluid contained in these feeds is about 30 oz., and the child requires about 36 to 40 oz. of water (at the rate of 2 to 2¼ oz. per lb.). This water can be given either as separate drinks or distributed over the four milk feeds, by adding 2 oz. of water to each feed to make it up to 8 oz. in bulk.

Diet for a Child aged 12 to 18 months (weighing say 23 lb.).—We shall now diminish not only the relative quantity of milk per lb., but also its absolute amount. We shall give only 28 oz. of milk. This will contain $28 \times 19 =$ about 530 calories. But the child requires $23 \times 45 = 1{,}035$ calories, therefore about 500 calories must be made up from other foods. We shall give one of the feeds of 8 oz. of milk in the form of a gruel made up in the proportions described above, and make up the balance with some egg, rusk, potato, vermicelli and butter. The gruel feed has the following composition:—

TABLE CXL.

Food	Protein	Fat	Carbo-hydrate	Calories
Gruel, 8 oz.	0·36 oz.	0·61 oz.	1·37 oz.	361
The 20 oz. of milk contain :—				
Milk, 20 oz.	0·70 oz.	0·70 oz.	1·00 oz.	380
We shall also give 2 oz. of rusk or biscuit.				
Rusk, 2 oz.	0·20 oz.	0·02 oz.	1·18 oz.	166
Total	1·26 oz.	1·33 oz.	3·55 oz.	907
We must therefore make up a balance of about 130 calories by means of egg, potato and vermicelli, as follows:—				
½ egg (= 1 oz.)	0·14 oz.	0·12 oz.	—	47
Vermicelli and butter (2 oz.) ..	0·06 ,,	0·06 ,,	0·34 oz.	62
Mashed potato (1 oz.)	0·02 ,,	—	0·18 ,,	23
Total per day ..	1·48 oz.	1·51 oz.	4·07 oz.	1,039

The quantities accord very well with what we found in Chapter XIII, p. 366, to be the requirements of protein, fat and carbohydrate at about this age, viz., 0·4 per cent., 0·4 per cent. and 1·2 per cent. respectively. Thus:—

TABLE CXLI.

	Protein	Fat	Carbo-hydrate	Calories
Theoretical requirements ..	1·47 oz.	1·47 oz.	4·41 oz.	1,035
Diet contains	1·48 ,,	1·51 ,,	4·07 ,,	1,039

In addition the child requires a total of about 46 oz. of water, but the diet only contains about 28 oz. of water (in the milk). Therefore it is necessary to give it another 18 oz. This can be given either as separate drinks or by mixing it with the milk feeds, as follows :—

7 a.m.—Gruel, 8 oz. ; a drink of water, 3 oz.

11 a.m.—Milk, 5 oz. ; water, 5 oz. ; ½ boiled egg ; 2 oz. of vermicelli in butter ; rusk, 1 oz.

3 p.m.—Milk, 5 oz. ; water, 5 oz.

7 p.m.—Milk, 5 oz. ; water, 5 oz. ; mashed potato, 1 oz. ; rusk, 1 oz.

TABLE CXLII.

	Protein	Fat	Carbo-hydrate	Calories
8 a.m.—Milk gruel, 6 oz. ..	0·27 oz.	0·45 oz.	1·07 oz.	271
½ egg (1 oz.)	0·14 ,,	0·12 ,,	—	47
A slice of toast, 1 oz.	0·11 ,,	—	0·61 ,,	83
Butter ¼ oz.	—	0·25 ,,	—	66
Total for breakfast ..	0·52 oz.	0 82 oz.	1·68 oz.	467
12 noon.—Sole 1½ oz., boiled in	0·28 oz.	0·02 oz.	—	38
milk, 1 oz.	0·07 ,,	0·07 ,,	0·10 oz.	38
Mashed potato, 1 oz...	0·02 ,,	—	0·18 ,,	23
A slice of bread, 1 oz.	0·11 ,,	—	0·61 ,,	83
Brussels-sprouts, 1 oz.	0·02 ,,	—	0 03 ,,	6
Baked apple, 1 oz. ..	—	—	0·10 ,,	12
Total for dinner ..	0·50 oz.	0·09 oz.	1·02 oz.	200
4 p.m.—Cup of cocoa, 8 oz. (milk, 8 oz., cocoa, ⅙ oz., sugar, ¼ oz.)	0·31 oz	0·37 oz.	0·72 oz.	217
Slice of toast and butter (as for breakfast)	0·11 ,,	0·25 ,,	0·61 ,,	149
Jam, ¼ oz.	—	—	0·17 ,,	20
Total for tea	0·44 oz.	0·62 oz.	1·50 oz.	386
8 p.m.—Milk, 4 oz.	0·14 oz.	0·14 oz.	0·20 oz.	76
Rusk, 1 oz.	0·10 ,,	0·01 ,,	0·59 ,,	83
Total for supper ..	0·24 oz.	0·15 oz.	0·79 oz.	159
Total for the day ..	1·70 oz.	1·68 oz.	4·99 oz.	1,211

Diet for a Child aged 18 to 24 months (weighing say 26 lb.).—We shall now add some fish or minced beef, as well as some vegetable, to the preceding diet, and it will be our task to arrange the dietary in such a way as to make the amount of protein, fat and carbohydrate, 1·66 oz. (= 0·4 per cent. of body weight), 1·66 (= 0·4 per cent.), and 4·98 oz. (= 1·2 per cent.) respectively, making a total of about 1,200 calories.

Table CXLII will be the dietary.

If we had substituted $1\frac{1}{2}$ oz. of beef for the $1\frac{1}{2}$ oz. of sole boiled in 2 oz. of milk at dinner, we would have given 0·33 oz. of protein and 0·37 oz. of fat for 0·35 oz. of protein and 0·09 oz. of fat. In other words, whilst the amount of protein would remain practically unchanged the amount of fat would become increased by 0·24 oz., and hence, to be strictly accurate in one's ratios as well as in total calories, one would have had to omit one of the rations of 0·25 oz. of butter either at breakfast or at tea.

Diet for a Child 2 to 3 years old (about 30 lb.).—At this age the child needs about 40 calories per lb. (total = about 1,200 calories), and the proportion of protein, fat and carbohydrate should be about 0·35 per cent., 0·35 per cent., or 1·05 per cent. of the body weight respectively, corresponding to 1·68 oz. each of protein and fat and 5·04 oz. of carbohydrate. Hence the diet suitable between 18 and 24 months will also serve during the third year.

Diet for a Child 3 to 4 years old (about 34 lb.). (Table CXLIII.)

On the basis of protein 3·25 per 1,000, fat 3·25 per 1,000, and carbohydrate 9·75 per 1,000 of body weight, the child requires protein 1·77 oz., fat 1·77 oz., and carbohydrate 5·37 oz.

After the age of 3 years the diet is gradually increased both in regard to quantity as well as variety and indigestibility. Thus lettuce, watercress, &c., may be added to the list of vegetables, and raw fruit (provided it is not too unripe) may be given without much risk. The following articles of diet, however, should not be permitted under the age of 6 years: Steak, onions, spices, salt beef, pork sausages, nuts, cheese, herrings, raw tomatoes, raw cucumbers, radishes, fresh bread, pastries, and excess of sweets.

The data given in this and in the preceding chapters will enable the reader to construct suitable dietaries at various ages. Fuller details, as well as tables giving the chemical composition of the various foodstuffs commonly used, will be found in books devoted to dietetics. (See Literature at end of chapter.)

TABLE CXLIII.

	Protein	Fat	Carbo-hydrate	Calories
8 a.m.—Milk gruel, 8 oz. ..	0·36 oz.	0·61 oz.	1·37 oz.	361
1 egg	0·28 ,,	0·24 ,,	—	94
Slice of bread, 1 oz.,	0·11 ,,	—	0·61 ,,	83
and butter, $\frac{1}{4}$ oz.,	—	0·25 ,,	—	66
and jam, $\frac{1}{2}$ oz.	—	—	0·35 ,,	41
Total for breakfast ..	0·75 oz.	1·10 oz.	2·33 oz.	645
12 noon.—Lean meat, 2 oz. ..	0·46 oz.	0·12 oz.	—	85
Vegetables—				
Potato, $1\frac{1}{2}$ oz. ..	0·03 ,,	—	0·27 oz.	35
Spinach, 1 oz. ..	0·04 ,,	—	0·05 ,,	10
Slice of bread and butter and jam, as before	0·11 ,,	0·25 ,,	0·96 ,,	190
Stewed fruit, 4 oz. (4 tablespoonfuls of apple sauce)	0·16 ,,	0·03 ,,	1·20 ,,	165
Total for dinner ..	0·80 oz.	0·40 oz.	2·48 oz.	485
6 p.m.—Small cup of cocoa, 6 oz.	0·23 oz.	0·27 oz.	0·54 oz.	162
Total for the day ..	1·78 oz.	1·77 oz.	5·35 oz.	1,292

The following sample diet sheet for a child 5 years of age is given by Eric Pritchard :—

TABLE CXLIV.

Breakfast	Dinner	Tea	Supper	Calories for 24 hours
Milk, 8 oz. .. Sugar, $\frac{1}{3}$ oz. .. Cocoa, $\frac{1}{4}$ oz. .. Bread or toast, $1\frac{1}{2}$ to $2\frac{1}{2}$ oz. .. Butter, margarine, or dripping } $\frac{1}{4}$ oz. Stewed fruit .. Baked apple .. Raw fruit, or lettuce	Beefsteak pudding, with plenty of gravy } 2 oz. Potato, 1 oz. .. Cabbage, 1 oz. .. Milk pudding, 2 oz. Stewed fruit, 1 oz. Bread, 2 oz. .. Water, 6 oz. ..	Milk, 8 oz. .. Sugar, $\frac{1}{4}$ oz. .. Cocoa, ovaltine, or chocolate } $\frac{1}{4}$ oz. Bread, toast, plain cake, oatmeal biscuits, shortbread } 3 oz. Butter, margarine, or dripping } $\frac{1}{4}$ oz. Jam or stewed fruit, or lettuce or watercress, or marmite	Soup or broth, 4 oz., or rice pudding, 3 oz.	
About 450 calories	About 350 to 500 calories	About 500 calories	About 150 calories	About 1,600 calories

General Rules with Regard to Diet.—The following rules should be observed by every child:—

(1) Wash the hands and face before and after meals.

(2) Eat slowly and chew the food well. A school child should get up sufficiently early not to bolt its breakfast in the hurry to get to school.

(3) The child must be trained to eat what is given it.

(4) Meals should be taken at regular intervals, and too many sweets should not be eaten, especially between meals, as this takes away the appetite for the proper quantity of the regular meal. Any sweets should be eaten directly after a meal.

(5) Drink between four and five glasses of fluid a day.

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CHAPTER XVIII

CLOTHING

"That the garments of infancy are largely governed in their design by age-long ceremonial cannot be doubted, and as with the dress of the soldier, the bride, the priest, or the judge, conformity, not convenience, comes to be the thing that counts."

Dr. Harold Waller.

The Ten Requisites of Children's Clothing are:—

(1) *Warmth.*—We have seen that in relation to the child's weight its surface area is much larger than in the adult, and also that the heat-regulating apparatus of the young infant is somewhat defective. For these reasons a child's underclothing must be made of material such as wool or flannel, that is a bad conductor of heat, so that it cannot easily carry away the heat of the body to the outside. It must be remembered, however, that in the same way as infants not sufficiently warmly clothed will lose heat rapidly and suffer from catarrhal inflammation of the respiratory or digestive tracts, so also will overclothing or exposure to excessive heat render them liable to similar trouble as the result of overheating of the body. Hence, in the heat of the summer it is important not only that children should not be overclothed, but that the material from which their clothes are made should be good conductors of heat, such as cotton or muslin. The same remarks apply to different climates. In parts of the world where the climate is cold and damp woollen shirts should be worn, but in dry, hot climates cotton shirts are more suitable.

(2) *Ability to absorb Moisture in Reasonable Amounts.*—Perspiration of the skin goes on continually, even in cool weather, as can be verified by the visible evaporation taking place when a vest which has been worn next to the skin for a few hours is held near the fire. The behaviour of different fabrics towards moisture depends upon four separate properties, viz.:—

(*a*) *Wetting property*, by which is meant the rate at which the material can imbibe water. In this respect

Harold K. Faber and Fannie Hadden[1] find that wool is the best material, linen is the next best, silk comes third and cotton fourth.

(*b*) *Total Capacity for Water.*—Wool and silk have the greatest capacity for water.

(*c*) *Rate of Evaporation of Absorbed Moisture.*—The material must not cause too rapid evaporation, as the loss of latent heat caused thereby might cause chilling of the body. It must also not be too slow an evaporator, or else injury to the skin, such as prickly heat or maceration leading to bacterial invasion, will result. When measured by the amount of water that can be evaporated per unit of surface per unit of time, it is found that canton flannel is the slowest, silk is the most rapid, whilst cotton and wool are intermediate.

(*d*) *Rate of Heat Conduction when the Fabric is Wet.*—Rubner found that whilst dry wool is the worst heat conductor, wet wool is the best conductor.

(3) *Permeability to Air and Ultra-violet Radiation.*—Any open-wove material is of course permeable to both. Cotton is five times more permeable to air than wool tricot. Of the other materials, Leonard Hill has shown that artificial silk, which is a cellulose material, is more transparent to ultra-violet rays than wool or silk, which are protein products. Hess found that whilst thin nainsook cotton will prevent a certain proportion of the rays from reaching the body, thick nainsook cotton will act as a barrier against a larger proportion of these rays. This property of permeability hardly arises, however, when more than one garment is worn.

(4) *Smoothness of texture*, so as to avoid irritation of the skin. Wool is the worst offender in this respect, although different skins are susceptible in different degrees.

(5) *Cleanliness.*—This property depends upon two factors, viz.:—

(*a*) *Ability to withstand efficient cleansing without rapid deterioration.* Wool, for instance, shrinks in the wash.

(*b*) *The rate at which dirt, &c., can be removed from the fabric by washing.* Faber and Hadden studied the behaviour of various materials in this respect by soaking equal pieces of material in cultures of *Staphylococcus*

[1] *Arch. Pediatr.*, May, 1926.

aureus (1 : 10), drying in an incubator, rinsing four times in sterile water and plating out 0·1 c.c. of the last wash and counting the colonies. They found linen to be the worst in this respect, giving over 22,000 colonies after the last wash, and silk to be the best, the number of colonies found being only about 1,400. There was no material that was rendered perfectly sterile by ordinary rinsing; boiling is necessary for this purpose.

(6) *Lightness*, so as not to throw any unnecessary weight upon the child, and thus interfere with the freedom of movement of the chest, abdomen and limbs.

(7) *Looseness*.—The clothes must fit the body loosely, but not untidily, for the same reason. Too tight clothing or binder may cause regurgitation of food.

(8) *Ease of putting on and taking off*.

(9) *Economy*.

(10) *Non-inflammability*.

Wool answers the purpose best, except from the point of view of cost as well as its tendency to shrink during washing. For this reason some poorer mothers make use of flannelette, which is warm and cheap. The great objection, however, against the use of flannelette is its *inflammability*, which is responsible for a considerable number of fatal burns in children every year. The same remarks apply to the use of celluloid (e.g., celluloid combs) which is also highly inflammable. The following simple experiment will illustrate the difference between flannel, flannelette and celluloid in this respect. Take a piece of each of these materials and apply a match to it. The flannel will be merely singed, but the other two pieces of material will burn up completely.

The shrinkage of woollen goods in the wash can be overcome, according to Jaeger, as follows: "The water should be warm, not hot, never above 100° F. Make 3 gallons suds with 1½ oz. best soap. Do not leave clothes to soak but work them about, pressing through the hands and *not rubbing*. Pass them through a wringer, and rinse twice in clean tepid water, passing again through the wringer. Stretch to shape and hang to dry. Iron when half dry, and stretch to proper size while under iron."

New woollen goods should be bought just a little larger than needed, to allow for some shrinkage.

When choosing coloured garments for children, it is well to remember that dark-coloured ones absorb more of the heat rays

of the sun than light ones and are therefore warmer; grey is an excellent tint for all seasons.

The amount of clothing children should wear varies with age, climate and season, but it is a fairly safe general rule that a few light woollen garments covering the body uniformly are sufficient.

Garments for Infants.—The following list of clothes answer the purpose well:—

(1) *Vest*, made of either wool or silk, or silk merino (fig. 110). The disadvantage of wool is the irritation it may cause to the baby's skin. This, however, may be overcome by dressing the baby in a shirt of some cellular material next to the skin, and over this to put on the woollen vest. The vest should be double-breasted and long-sleeved.

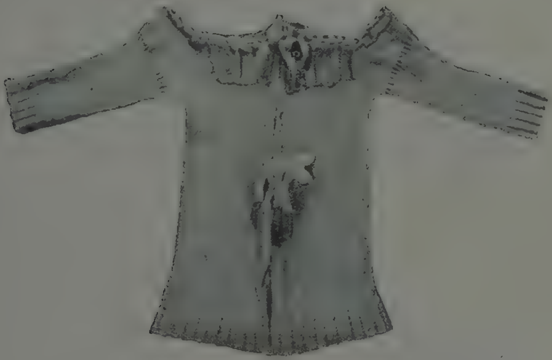

FIG. 110.—Vest.

(2) *Diaper.*—Harrington's absorbent napkins are, according to Mabel Liddiard, Matron of the Mothercraft Training Society, soft, easy to wash, dry, and in every way ideal for the baby. Outside this, one made of Turkish towelling should be folded round as a pad. If the diaper is pinned to the vest, one must be careful not to pin it too high, since any undue tension on the shoulders may produce deformity. In any case, no other than safety pins may of course be used. It is better, however, to tie the napkins with tapes sewn on to them instead of using safety-pins. The diaper should be applied loosely enough not to interfere with the movements of the legs, and should be removed as soon as soiled, since soiled diapers may cause cystitis and ascend-

ing pyelocystitis. Also, unless handled with the greatest care, they may be a potent agent in the spread of summer diarrhœa (see p. 158). Napkins should not be washed in soda, which may irritate the skin.

(3) *Woollen drawers or a pilch* over the diaper (fig. 111).

(4) *Petticoat* (fig. 112), of loosely-woven (and therefore permeable to air) flannel, of about 23 or 24 in. in length, so as to cover the feet. It can be tied with tapes at the foot if desired. This garment is now used instead of the *long* robes that had been the fashion for years, and which used to be turned over the baby's legs to keep them warm.

Fig. 111.—Drawers.

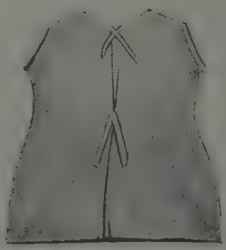

Fig. 112.—Petticoat.

The *short clothes* have the advantage over the long ones in that:—

(*a*) They are cheaper, because they use up less material and need not be discarded when the infant gets older.

(*b*) They take less time in making and in washing.

(*c*) They afford more freedom of movement to the legs.

(*d*) They are not so heavy.

(*e*) They make it easier for the nurse to train the baby into good habits.

(*f*) They allow free movement and exercise of the lower limbs.

(5) *Frock* (fig. 113), just long enough to cover the petticoat, and made of cashmere, flannel, soft silk, or similar material.

(6) *Coatee*, made of cashmere or wool, to be worn over the frock in cold weather.

(7) *Bootees*, knitted or crocheted in wool, and reaching to the knees are needed to keep the feet and legs warm, especially in cold weather, as cold feet are dangerous in delicate babies.

In the summer thin silk socks and soft kid bootees may replace the woollen bootees.

(8) *Cap*, which should not be heavy enough to produce perspiration, is advisable, either in very cold weather to avoid the risk of catching a cold, or in very hot weather to protect against sunstroke. Soft muslin is the best material for this.

(9) *Mittens*, woollen or fur, for winter outdoor wear.

Veils have the disadvantage that they prevent the escape of impure expired air as well as the intake of pure fresh air. On the other hand, in the hot season they are useful as a protection against flies.

FIG. 113.—Frock.

A **binder** round the abdomen is only necessary during the first week to keep the cord dressing in place. After the cord has fallen off the binder should be dispensed with, for the following reasons :—

(1) It is not necessary for the purpose of protecting the internal abdominal organs against chill, because the woollen garments do this quite efficiently.

(2) If applied loosely, it very soon slips up and encircles the chest rather than the abdomen, preventing its normal respiratory movements.

(3) If applied tight enough to keep in place it compresses the abdomen, and not only impedes its normal respiratory excursions, but it may also cause discomfort because it does not allow for any intestinal distensions in the process of digestion.

(4) By supporting the back it weakens the spinal muscles, and this may result in spinal curvature when the baby has to sit or stand up and support its back of its own accord.

The baby or child, as we have said, requires to have all its clothing loose so as to allow free play for all its muscles. Freedom of movement of the thorax develops the intercostal and other respiratory muscles and thus encourages free expansion and aeration of the lungs. The same applies to freedom of movement of the legs, arms, head, &c.

Garments for Older Children. — The garments of older children should be so made as not to interfere with the free play of the limbs or other parts of the body. As soon as the child begins to run about its feet must be shod with the greatest care. The **shoes** should be light and wide enough not to compress the toes and to allow for growth of the feet. On the other hand, they must not be too wide to rub and produce blisters on the feet. A child's feet are generally well formed at birth and only become distorted from the use of unsuitable covering. In infancy the big toe is separated from the others, and a properly fitting shoe or boot should prevent this toe from being pushed outward. The malposition of the great toe, which is invariably caused by the constant pressure of ill-fitting footgear, is usually the cause of corns and bunions and many other evils. The soles should be thick and flexible to protect the foot against cold and damp and flexible to give spring to the foot. They should, during the first few years, be practically flat; but low, non-tapering heels may be used after the fifth year. Shoes are better than boots because they allow more freedom of action of the leg muscles. Sandals and bare legs are nice for the hot summer days, but when the cold weather comes stockings and shoes become a necessity.

The **stockings** should preferably be of the one-toed shape since when sandals are worn they allow the strap to pass between the big toe and its neighbour.

The remainder of the little **boy's outfit** should consist of light woollen combinations (fig. 114), a suit over it of short trousers made of light all-wool serge buttoned on to a liberty bodice, and a jersey or sailor blouse (figs. 115 and 116).

In the summer, if all-wool serge or cashmere is worn, there is really very little need for anything else, provided the clothes are frequently washed and kept very soft and pliable. All the

FIG. 114.—Combinations.

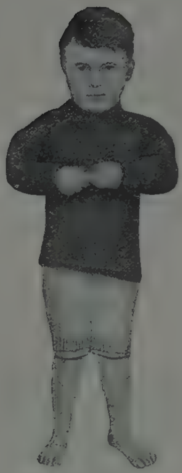

FIG 115.—Sailor blouse.

FIG. 116.—Jersey.

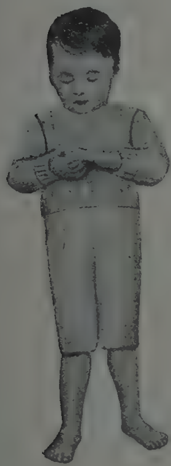

FIG. 117.—Bodice and bloomers.

FIG. 118.—Bloomers.

garments, including the trousers, must be wide enough to allow of perfectly free movements.

A **great coat** should be made of rain-proof, light and warm material, and should have ample room across the chest and under the arms so that the child can swing his arms round without any discomfort.

For little **girls**, the boots, shoes, stockings and combinations may be the same as for boys; a light woollen bodice over the combinations with buttons at the side (fig. 117), bloomers

Fig. 119.—Kilted skit and sailor blouse.

Fig. 120.—Pinafore dress.

Fig. 121.—Pyjamas.

(fig. 118), or kilted skirt made of thick or thin woolly material according to the time of year, button on the bodice. A sailor blouse or jersey goes well with a kilted skirt (fig. 119), or, if preferred and it is certainly the coolest for the summer—an overall or pinafore dress (fig. 120) is both pretty and cool. In hot weather the over bodice should be of the thinnest material; the combinations should be of gauzy woollen material and quite loose.

Night Clothes.—For sleeping, pyjamas of light woollen material, all made in one, are by far the best (fig. 121). Some pyjamas are made with feet, but the footless ones are most

hygienic, as they allow of ventilation. If necessary, woollen bed-socks can be worn for cold feet.

The Common Errors in Clothing Children are :—

(1) The use of too many and too heavy clothes. These to a large extent abrogate two of the functions of the skin, viz., to act as an accessory to the lungs in the process of respiration, and of the kidneys in the process of elimination of water and excretory products. When the child is overclothed the skin is deprived of each of these functions and extra work is thrown upon the lungs and kidneys, with harmful consequences to each of these organs.

(2) Wearing the same clothes by day and by night, so that no opportunity is given to the clothes to be aired and get rid of the absorbed moisture (see p. 456).

(3) Lack of warmth to the feet, which is a potent cause of "colds." The soles of the boots should therefore be not too thin.

(4) The use of too tight clothes (see p. 458). Girls used to wear corsets with the idea of supporting their backs; the fact is, however, that such appliances, by not giving the muscles of the back sufficient work to do, weaken these muscles, so that the back cannot be kept straight when the artificial supports are removed. With the growth of physical exercise for children corsets have disappeared. Tight garters, which interfered with the circulation in the lower extremities, have equally been displaced by suspenders. Tight shoes cause corns and distortion of the feet, and are also responsible for cold feet and chilblains.

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CHAPTER XIX

MUSCULAR EXERCISE

"Muscles are in a most intimate and peculiar sense the organs of the Will. They have built all the roads, cities and machines in the world, written all the books, spoken all the words, and, in fact, done everything that man has accomplished with matter. If they are undeveloped, or grow relaxed and flabby, the dreadful chasm between good intentions and their execution is liable to appear and widen. Character might be in a sense defined as a plexus of motor habits. . . . Muscle culture develops brain centres as nothing else demonstrably does."

STANLEY HALL.

THE value of bodily exercise has been appreciated ever since the time of Hippocrates (460-370 B.C.). The Father of Medicine writes: "The untrained have moist flesh, and when they exert themselves the body becomes heated, and they yield the product of liquefaction in abundance."

He also recommends abdominal kneading for constipation.

The education of the Spartan child of either sex was almost entirely physical: Scouting, riding, hunting, swimming, boxing, wrestling, horse-racing, &c., and in the case of girls, also dancing to music. The children were, at the age of 7 years, put in a boarding school where their individual peculiarities were rubbed off by their contests with their schoolmates. At Athens children stayed at home and went to school daily, but exercise in the open air was indulged in for several hours a day. Similar exercises were practised by Jewish children in the times of the Talmud.

The first writer who lays down a definite programme of *exercises for young infants* was the Greek physician, Soranus of Ephesus, of the second century A.D. We have already given full details of those exercises in Chapter II (p. 36), but their importance at such an early age seems to have been hitherto overlooked by those who were in charge of young babies. As we shall see, the exercises that have of recent years been devised and recommended for babies are practically identical with those described by Soranus. George Armstrong, in a book published in 1777 ("An Account of the Diseases most Incident to Children, from

Birth till the Age of Puberty"), recommends massage for very young infants twice a day in front of a fire. "There is nothing," writes Armstrong, "that infants in general seem more delighted with than this exercise, and it were to be wished that the nurses would indulge them more in it . . . it is not only pleasing to them, but conduces greatly to make them thrive, and to prevent their catching cold, by promoting a free circulation, and perspiration likewise; Providence having kindly connected the agreeable sensation and the benefit."

Francis Glisson, more than a century earlier, describes certain exercises for the treatment of rickets, such as holding the child gently "under the Arms, yet so that the Thighs and the whole Body may hang down. Also the drawing of the Children backward and forward upon a Bed or a Table between two Nurses, the one holding it by a Hand, the other by a Foot." . . . He also speaks of "the artificial suspension of the Body" by means of swathing bands passed under the Armpits, "so that it is a pleasure to see the Child hanging pendulous in the Air," as a useful exercise "to restore the crooked Bones, to erect the bended Joynts, and to lengthen the short Stature of the Body."

Bartholomaeus Metlinger, of the fourteenth century, prescribes exercises after the infant's bath. "After the bath, before the child is wrapped up, the legs should be bent backwards, and also the feet brought forward to the head, especially in boys, so that their joints will be more supple. The joints should be softly rubbed, and if there is anything to stretch it should be stretched . . . and if the child has misshapen joints or limbs, as Galen tells, they should be anointed with rose oil after the bath and placed in the best position, and if necessary, they should be bound up."

Physiology of Exercise.—The execution of any voluntary muscular movement involves the activity of the following parts: (1) The brain where the impulse is initiated, (2) nerve fibres which transmit the impulse from the brain to the muscles, and (3) the muscles which, by contracting, perform the movement. Hence every voluntary movement depends upon the integrity of these three different parts. Now when any muscle contracts it does work and uses up energy, and therefore an increased supply of nourishment must be carried to it by the blood. Also, in the process of contraction some portion of the muscle tissue is burned up, and the waste products thus produced must be removed by the blood. These waste products, if present in the blood in

normal amounts, stimulate the various "centres" in the brain which control the action of the heart and of the lungs.

We thus see that exercise has not only a local effect upon the muscles themselves but also influences the activity of the other systems in the body. We shall consider some of these effects in greater detail.

(1) *Effect upon Muscles.*—The additional nourishment brought by the blood to the muscles supplies them with extra nourishment, so that muscles regularly and suitably exercised become larger and stronger.

(2) *Effect upon Bones.*—The bones to which the exercised muscles are attached also benefit by the increased local blood-supply. The skeleton therefore becomes stronger and is capable of bearing greater strain.

(3) Similarly the *joints*, in virtue of the repeated movement, become stronger and more flexible.

(4) *Effect upon the Circulation.*—As we have seen, the waste products from the muscles stimulate the centre controlling the circulation and make the heart beat more rapidly in order to send an extra supply of nourishment to the muscles. Also the contraction and relaxation of the muscles alternately empty and refill the veins inside them, thus helping the venous circulation. In this way the muscle of the heart itself is rendered stronger and healthier.

(5) *Effect upon Respiration.*—The muscular waste products also stimulate the centre in the brain controlling respiratory movements, and as a consequence the breathing becomes deeper and more rapid. More oxygen is therefore breathed into the lungs and more carbonic acid gas is breathed out.

(6) *Effect upon the Excretory Organs*—

(*a*) The effect of exercise upon the excretion of CO_2 from the *lungs* has just been stated.

(*b*) *Skin.*—As the result of the accelerated action of the heart the blood-vessels in the skin become filled with blood, and perspiration also becomes increased. Both the flushing and the sweating of the skin result in a loss of heat by means of radiation and evaporation, and in this way the body gets rid of the extra heat produced by the muscular contraction, as well as, by means of the sweat, of impurities resulting from the burning up of the muscle tissue.

(*c*) *Kidneys.*—The kidneys also help to eliminate the waste products by means of the urine.

(7) *Effect upon the Digestive System.*—Muscular exercise increases the appetite and therefore aids nutrition.

(8) *Effect upon the Nervous System.*—In addition to the part taken by the nervous system in the execution of every voluntary muscular contraction which I have already described, there is still another relationship between the contraction of a muscle and the nervous system. Each muscular contraction sends up a stimulus, by means of certain sensory nerve paths, which inform the higher centres not only of the degree of contraction but of the position of the joint which is moved, in space as well as the amount of resistance overcome. In this way not only does frequent repetition of any muscular act help to establish firmly these nervous paths, but their effects are stored up in the brain as a permanent memory, rendering the future execution of such acts easy and automatic. Moreover, the concentration that each exercise requires, as well as the discipline that it demands of the child to respond promptly to the word of command, makes physical exercise a valuable training in alertness, accuracy and precision. It will therefore be clear that the active use of muscles is of very great importance, not only from the point of view of muscular development and general organic health of an individual, but also in regard to the development of that sensory and emotional control which is so essential to the formation of character.

The influence of sensory nerve paths on voluntary muscular contraction is seen when these paths are thrown out of action, experimentally or clinically, when, in addition to the lack of stability, symptoms of paralysis arise as the result of loss of sensation in the part in spite of the integrity of the motor paths.

It is especially during infancy and childhood that the beneficial influence of properly arranged and suitably graded exercises makes itself felt. For, during these periods of growth the body as well as the mind are still in a very plastic condition, and are extremely susceptible to the effects of stimuli. The nurse or teacher must realize, however, that the infant or child under their care is a very delicate organism, and the kind of exercise which is suitable for a child of one particular age is not suitable for one of a more tender age.

The Peculiarities of the Child's Muscular System.

The following are some of the more important differences between the muscular systems of the child and of the adult :—

(1) The relative weight of the muscles is less. Thus, in the

newborn child the muscular system is about 23 per cent. of the total body weight compared with 43 per cent. in the case of the adult.

(2) Chemically, the child's muscle contains a greater proportion of water. At birth, for instance, the percentage of water is about 82 per cent., whilst at 2 years of age it is only 77 per cent. In the adult muscle water is present in still smaller percentage. The amount of myosin also, upon which the contractility of a muscle depends, is less in the child, it being 5 per cent. in the newborn infant and 6·4 per cent. at 2 years of age.

(3) The area of cross-section of each individual fibre as well as of any particular muscle as a whole—upon which the strength of a muscle depends—are less in the child than in the adult. The same applies to the total number of muscle-fibres in any muscle.

(4) *The force per unit area* of muscle is, however, apparently the same at all ages.

(5) The infant's muscles are more prone to fatigue than those of the adult.

(6) The child's muscles are not so easily or delicately co-ordinated as those of the adult.

"On the laws of muscular development," says Terman,[1] "if the related facts were fully at our command, an entire philosophy of education could be based." The order of development of individual muscles or set of muscles is of great importance from the hygienic standpoint. The fact that the control of the accessory muscles, such as are used in carrying out the finer movements of drawing, piano-playing, &c., develops later than that of the fundamental muscles is obviously of practical importance in manual and industrial training, writing, gymnastics, &c. Thus, play, games, and manual exercises which demand on the part of the child the delicate co-ordination of accessory muscles (e.g., of the fingers and hands) are unsuitable for infants under 3 or 4 years of age.

Fatigue.—Since muscular movement involves activity on the part of the brain-cells, nerve-fibres and muscle, it is clear that the cause of fatigue—when the muscle refuses to respond readily to stimuli—may reside in any one or more of these parts. It has been shown experimentally, however, that it is the nerve-

[1] Lewis M. Terman, "The Hygiene of the School Child." London, 1913, p. 53.

cells in the brain, as well as the nerve-endings in the muscle, which become tired long before the muscle-fibres show signs of fatigue, and that the conducting nerve-fibres never become fatigued at all. It is possible in a "muscle-nerve" preparation to paralyse the nerve-endings in the muscle by means of curare. If now the nerve be stimulated for hours, the impulse cannot reach the muscle-fibres. If the effect of the curare is allowed to pass off (whilst the nerve is still being stimulated) the muscle begins to contract, showing that though the nerve was continually being stimulated all the time there was no fatigue manifest in it. Similarly, if a muscular movement be repeatedly made by continuous electrical stimulation of either the particular centre in the brain or of the motor-nerve until the muscle refuses to respond, it is found that direct stimulation of the muscle-fibres at once results in a contraction. From these experiments we see that the cerebral cortex as well as the nerve-endings tire before the contractile power of the muscle is lost through fatigue. This fatigue is brought about by the action of the poisons formed as the result of the disintegration of muscle-tissue being circulated in the blood, and bathing the nerve-endings in the muscle as well as the cerebral nerve-cells. In this sense, therefore, the muscles may be said to be "the slaves of the nerve-cells" (Tait McKenzie), since the will tires long before the contractile power of the muscle gives out. If the muscle is allowed a sufficient period of rest the production of waste matter ceases, and the poisons which have accumulated are gradually eliminated by the lungs, skin and kidneys, and fatigue passes off. If, however, the exercise is continued before the poisonous products are eliminated, not only are the muscles and nerve-centres continually subjected to the poisoning action of these toxins, but the whole body becomes affected and suffers in consequence. The younger the child the more easily it gets tired, and therefore extreme care is necessary on the part of the nurse or teacher not to over-fatigue the child.

It is to be remembered, however, that muscular exertion will produce less fatigue in a child that is well nourished, that has a sufficient amount of sleep, and that is living under good hygienic conditions with regard to fresh air, sunshine, &c., than in a child living under less favourable conditions. It is in this latter class of children that a small accumulation of toxin is sufficient to produce the phenomena of fatigue. This teaches us that as soon as an infant begins to show displeasure at the exercise, or the

school child exhibits a failure to respond, or inaccuracy in the execution of the allotted tasks, it must be given at once a period of rest.

That fatigue is produced by the waste products of muscular contraction has been experimentally proved as follows. An animal is experimentally fatigued and immediately afterwards killed. An extract is made from its muscles and injected into the circulation of another *fresh* animal, when the latter begins to show evidence of fatigue. Control experiments made with extracts of unfatigued muscles give no such results.

Exercise during Infancy.

Every infant possesses the instinct of spontaneous muscular movement, and at the very earliest age this play instinct should be allowed to develop.

The infant should from birth be allowed free movement of its limbs, to kick with its legs and to pull or grasp with its hands. When it is a few weeks old it should be placed naked on the bed, or on a warm rug on the floor in a well-warmed room, and allowed to kick against something, or to pull with his hands some such object as a ring or a handkerchief. In this way the pushing reflex as well as other bodily reflexes will be developed. It should be rolled over and allowed to lie on its abdomen. In this position the desire to see things around it makes it raise its head, thus helping the neck muscles to develop. When the ability of raising its head has been acquired, the infant will try to raise its thorax and support itself on its arms, thus exercising and strengthening its spinal, pectoral and brachial muscles. In the prone position, too, the infant soon makes attempts at crawling, with the consequent strengthening of its abdominal and leg muscles, thus preparing it for the acts of standing and walking.

The movements of the arms begin to be co-ordinated at about three to four months, and the baby is then able to grasp objects. At that time it is necessary for the mother or nurse to see that the child does not put any small objects into its mouth, lest they find their way into the respiratory passages, with serious consequences.

On account of the delayed myelinisation of the nerve fibres supplying the lower limbs, the co-ordinated movements necessary for walking are not as a rule developed till the child is one year old. Walking, however, commences somewhat later in boys than in girls, and in fat than in muscular children. *Rickets*, *anæmia*, *general debility*, *such as that following an acute illness*, *eruption*

of a tooth, &c., delay the onset of walking. If the child does not walk when it is 18 months old, it should be medically examined for rickets, brain disease (imbecility, Little's disease, &c.), cretinism, congenital idiocy, congenital dislocation of hips, &c.

Crawling.—If the infant has been accustomed to lie on its abdomen, crawling soon comes as a matter of course. This quadrupedal mode of progression is an excellent preliminary to walking, since all the muscles which are used in walking are here exercised and developed, gradually and gently; for the weight of the body, instead of falling on the two legs as it does in walking, falls on all the four limbs in crawling, and, therefore, the legs only have half of the body weight to support. As these muscles develop the child soon begins to stand up for a few seconds, when it comes against any object against which it can support itself, and soon after that it begins to walk a few steps, supporting itself against any object that happens to be handy.

The act of crawling is not only a very good preparation for walking, but it also gives free lateral mobility to the spine; with each forward push of the leg or arm the spine bends towards that side, and when the limbs of the opposite side move, the spine moves to the other side. This free lateral mobility of the spine in crawling is an excellent safeguard against early lateral curvature. Indeed, Spitzy[1] even recommends crawling therapeutically as a mode of correcting spinal curvature in children.

When the baby is able to crawl, it should be encouraged to climb a step a few inches high and be allowed to fall off the step on to a soft pillow. This will train the baby in self-control.

Walking.—After the child has learnt to crawl, and has made several attempts to stand up and walk a few steps by the aid of some support, the walking muscles and bones have become stronger, and the necessary nervous paths between the higher centres and the locomotor muscles have become established. When this has happened the frequent repetitions of the act soon render it automatic, in the same way as frequent practice at the piano ensures the player an automatic movement of the fingers so that he can play without either looking at the keyboard or thinking of what he is doing. When the child begins to walk, it often stretches out both arms in front and also swings its spine laterally in order to maintain its equilibrium forward and from side to side in a manner similar to that of the rope walker.

[1] Rachitis und Frühskoliose, *Zeitschr. f. orthop. Chirurgie*, xiv.

Parents have a natural eagerness to see their babies begin to walk, and often try to assist them by holding them with their hands under the babies' armpits. This is a practice which is not to be encouraged on account of the fatigue which it causes to the baby without hastening the regularity of such walking movements. It is better to rely upon the baby's natural instinct and its faculties of observation and imitation. The special apparatus for teaching babies to walk are similarly unnecessary and probably harmful. The best way of allowing a baby to teach itself the art of walking is to allow it to crawl.

It is advisable when the infant first begins to walk to protect its head against unnecessary injury by putting on it a soft head pad or tumbling cap.

FIG. 122.—A baby gripping the fingers.

The first movements are awkward and irregular, but this irregularity disappears at about two years, although it is still unable to run properly till three years old.

Special Exercises.—In addition to allowing the baby freedom to use the spontaneous exercises of its own devising, its natural play instinct should be aided and encouraged by a series of systematic exercises. The exercises prescribed by Soranus, of Ephesus, nearly 2,000 years ago (see p. 36), have recently been revived by Army officers (e.g., Muller in this country, and Neumann-Neurode in Germany). According to Professor Langstein, who had these exercises tried in his clinic in Berlin, they are so beneficial that they give rise to a better constitution and finer poise of the body when the infants grow up.

These exercises should be performed in a warm room with the baby undressed, and in the summer near an open window. They should not be done soon after a bath or on a full stomach nor when the baby is not quite well. As soon as the baby shows evidence of fatigue, such as displeasure or crying, the exercises must be stopped. During the first few weeks of life, massage

FIG. 123.—(J. P. Muller).

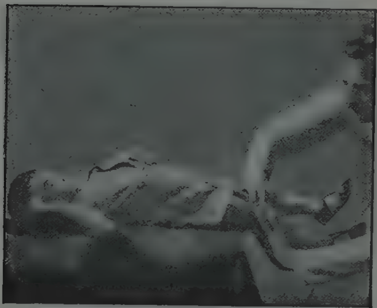

FIG. 124.—(J. P. Muller).

alone, i.e., length and cross rubbing of the whole body, is sufficient. Afterwards the following exercises may be carried out :—

Exercise 1.—Arm movements, resembling those of Sylvester's method of artificial respiration (fig. 123).

Exercise 2.—Leg movements. Stand in front of baby and grasp the legs gently in each hand, and move the limbs at the various joints, first very slowly, then at walking rate. Also practise circumduction (fig. 124).

Fig. 125.—(J. P. Muller.)

Fig. 126.—(J. P. Muller.)

Fig. 127.—(J. P. Muller.)

Exercise 3.—Respiratory movements. With baby lying on its abdomen, stand at the side of it, grip the bottom of its chest between your palm at the back and thumb and fingers at the sides, and practise light compression and relaxation at the respiratory rate (fig. 125).

Exercise 4.—Spinal Exercises. Place the infant on its belly with head on the side, and by encircling its legs in your hands raise the baby until it is nearly vertical (head down). The direction of the head should be changed frequently (fig. 126).

Exercise 5.—Gentle rotatary movements of spine (fig. 127).

FIG. 128.—(Neumann-Neurode.)

Exercise 6.—Exercise of spine by putting your hands into baby's axillæ (baby lying face down, and you standing at its feet) and raising its trunk gently.

Exercise 7.—Raising. Lay baby face down. Stand at its feet, grasp its hands and raise baby gently (fig. 128).

Exercise 8.—Lay baby's neck (face upwards on your right wrist, and hold up its ankles with your left hand. This teaches baby to keep its spine straight by its own efforts (fig. 129).

Exercise 9.—Leg and abdominal work. Raise baby's legs (baby lying on back) up to nose and let them go. Baby will gradually do this by itself (fig. 130).

FIG. 129.—(Neumann-Neurode.)

FIG. 130.—(Neumann-Neurode.)

Exercise 10.—Baby lying face down, push it gently by its legs. It will start making crawling movements (fig. 131).

Exercises for Older Children.—Till the age of 6 or 7 years children spontaneously exercise their muscles by running, skipping, playing with hoops and similar exercises. Such movements strengthen their muscles and render their joints pliable.

After the age of 7 years the child is able to do some more strenuous exercises. **Swimming** is an accomplishment which every child should be taught well, so that the child feels just as much at home in the water as on terra firma. This not only exercises every system in the body including equilibration, but is useful from the point of view of life saving.

Fig. 131.—(Neumann-Neurode.)

Bathing and swimming should not be done within two hours after a meal, and the child should never stand still in the water. The duration of a bath varies with the temperature of the water, and of the air, as well as with the particular idiosyncrasy of the child. The best guide is the sensation of the child. As soon as the child feels chilly he should leave the water and rub himself thoroughly with a bath towel, and walk and run about until he feels warm.

Cycling is another very useful exercise in muscular co-ordination and balancing, and can be begun at the age of about 5 years.

Climbing and jumping, as well as the usual games commonly played by children, are all useful.

Scouting is a most valuable training in methods of self-reliance and of overcoming difficulties, and helps to develop self-control, unselfishness, and a spirit of initiative, and should be undertaken by every child after the age of 7 years.

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CHAPTER XX

THE RÔLE OF SUNLIGHT AND ITS ARTIFICIAL SUBSTITUTES IN CHILD HYGIENE

"I marvel not, O Sun! that unto thee
In adoration Man should bow the knee,
And pour the prayer of mingled awe and love;
For, like a god thou art, and on thy way
Of glory sheddest with benignant ray,
Beauty, and life, and joyance from above."

—SOUTHEY.

HELIOTHERAPY, or the use of sunlight for healing purposes, dates from the times of early antiquity. Sun-baths were recommended by Hippocrates, Celsus, Galen, the Talmudic Rabbis, as well as by the ancient Egyptians. According to Cicero and the Younger Pliny (A.D. 40-50) solaria were built on the villas of the richer citizens of Rome. The Bible speaks of "the Sun of Righteousness . . . with healing in his wings," and the Talmud has a proverb to the effect that "When the Sun appears the patient recovers." Juvenal expresses a similar idea in the words, "Spes vitæ cum sole redit." Each of these is the analogue of the modern saying that "Where the sun enters the doctor never does." The practice of heliotherapy, however, fell into disuse until 1774, when Faure treated ulcers by exposure to the sun. Two years later, Ley Peyre and Le Comte focused sunlight on to wounds with good results. Cauvin, in 1815, advised parents to send their scrofulous and rickety infants to the country to be exposed to the sun. In 1859 Charcot showed that the effect of light rays on the skin was independent of any heating action. An important paper by A. Downs and P. Blunt appeared in the *Proceedings of the Royal Society*, in 1877, on the "Action of Actinic or Ultra-Violet Rays on Bacteria," and Robert Koch, in 1890, showed that the tubercle bacillus was killed by light. Downs and Blunt also showed that this bactericidal power is removed if a glass screen is interposed between the light and the bacilli. The pioneer in the domain of heliotherapy, as applied to child hygiene, is Dr. Theobald Adrian Palm, who, in 1890,

published a paper in *The Practitioner* on "The Geographical Distribution and Ætiology of Rickets," in which he pointed out the relation of rickets to darkness and sunlight, and urges "the systematic use of sun-baths as a preventive and therapeutic measure in rickets and other diseases." Professor Finsen, of Copenhagen, in 1893, published the results of sunlight treatment in certain tuberculous disorders, and in 1902 J. H. Sequeira introduced the treatment into the London Hospital. In 1903 Rollier opened his now world-famous Institution for Heliotherapy, at Leysin, for the treatment of surgical tuberculosis, and in 1910 he started his first École au Soleil, at Cergnat, near Leysin, for pre-tuberculous children. In England, work similar to that of Rollier has been carried out by Sir Henry Gauvain at Alton and Hayling Island. Since then works on the science and art of heliotherapy have appeared throughout the world. In this country Professor Leonard Hill and his co-workers (Argyll Campbell and Albert Eidinow) have investigated the subject experimentally.

Percy Hall, F. H. Humphris, Drs. Eleanor H. and W. K. Russell, and others have studied the subject clinically, whilst Dr. C. W. Saleeby has been ceaseless in his propaganda work, both by means of his fertile pen as well as his eloquent tongue, in pointing out the evils of darkness and the beneficent effects of sunlight. In December, 1924, the Sunlight League first published its journal, called *Sunlight*. This League, of which Queen Alexandra was the first patron, has for its objects the abolition of smoke and slums, and the education of the public in the value of sunlight in the prevention of disease. The *British Journal of Actinotherapy* first appeared in March, 1926.

The Physics and Physiology of Sunlight.

The sun and the numerous stars, in the process of their combustion which renders them self-luminous, pour out into space vast amounts of radiant energy in the form of periodic vibrations through the ether. These oscillations consist of waves of different lengths, as measured from peak to peak. The proportion of this radiant energy which reaches the earth is probably no more than one "two-thousand-millionth" part ($0{\cdot}5 \times 10^{-9}$) of the total radiation emanating from the sun. All these vibrations travel through space at the same speed of about 186,000 miles (300,000 kilometres) a second, although not

all have the same wave-length. Some of these, like the Hertzian waves used in wireless telegraphy and broadcasting, have wave-lengths between 3 mm. and several kilometres long, whilst others, like the gamma rays, have wave-lengths which are known to be no longer than one "hundred-million-millionth" of a millimetre, or one "hundred-thousand-million-millionth" of a metre (10^{-17} metre). The visible rays, or those which we recognise as light, have waves intermediate in length between the two extremes, though they approach much nearer the smaller extreme. The number of waves propagated per second is obviously equal to 186,000 miles, divided by the wave-length of the ray.

That white light consists of a number of rays of different colours was first proved in 1672 by Newton, who broke up a beam of light by means of a glass prism into its various colours, ranging between red and violet. In 1800 Herschel found that a thermometer placed outside the red of the spectrum showed a rise in temperature, and thus demonstrated the infra-red region. The existence of rays beyond the violet end of the spectrum was first noted by Scheele in 1777, and later by Ritter and Wollaston, in 1801, by the fact that silver chloride placed outside the beam of violet light became blackened. In this way the existence of chemical or actinic rays of the ultra-violet region was demonstrated. It is these rays which have been found to possess such powerful and valuable therapeutic properties (see p. 494), and treatment by means of them is called *actinotherapy*.

We see then that in addition to the visible spectrum there are two other regions, which cannot be perceived by the eye, possessing important properties, viz., the infra-red and the ultra-violet regions. Each of these regions consists of numerous rays of different wave-lengths and possessing different properties. (Compare the very high-pitched and the very low-pitched sound vibrations which cannot be perceived as sound by the ear.)

The following are the units in terms of which wave-lengths are measured. It will be readily seen that each of these units is some integral or decimal multiple of the other :—

TABLE CXLV.

Unit	Symbol	Millimetres
Ångstrom	Å or A.U.	One ten-millionth
Millimicron	$M\mu$ or $\mu\mu$	One-millionth
Micron	μ	One-thousandth

TABLE CXLVI.—GIVING THE WAVE-LENGTHS AND PROPERTIES OF THE VARIOUS REGIONS OF THE ELECTRO-MAGNETIC SPECTRUM.

Radiation and properties of electro-magnetic waves	Wave-length	
	Å	μ or $\mu\mu$
Ultra-violet	0-3,900	0-390 $\mu\mu$
Extreme region	0-2,000	0-200 ,,
Gamma rays (emitted by radium) (6 octaves)		
Röntgen (X) rays (14 octaves) (emitted by vacuum tubes)	0·1-500	0·1-50 ,,
Middle region	2,000-3,000	200-300 ,,
Near region	3,000-3,900	300-390 ,,
Visible light (1 octave)		
Violet to red	3,900-7,700	390-770 ,,
Infra-red (9 octaves)..	..	0·77-∞ μ
Near region	..	0·77-20 ,,
Infra-red (photography)	..	0·77-1 ,,
Rays passing through fluorite prism ..	..	1-10 ,,
Rays passing through rock-salt prism ..	..	10-20 ,,
Intermediate region	..	20-500 ,,
Extreme region (28 octaves) (Hertzian waves, &c.). Some of these are many kilometres long and have a frequency of only 10,000 per second	..	500-∞ ,,

As M. Guilleminot writes: "Science has put an immense keyboard under our fingers, but of this heliotherapy only makes use of a few notes."

Whenever one speaks of waves, whether of aerial vibrations constituting sound, or ether vibrations constituting the electro-magnetic spectrum, one speaks of one wave being an octave higher than another when the wave-length of the one is half that of the other (or, which is the same thing, when the rate of vibration or frequency of the one is twice as rapid as that of the other). White light consists of no more than one octave of vibrations, since the wave-length of the red rays at one end (7,700 Å) is about twice that of the violet rays at the other end (3,900 Å). The whole range of electro-magnetic waves extends over 60 octaves. Indeed, the visible portion of the spectrum constitutes only a minute portion, no more than one "ten-thousand-millionth" part of the total known electro-magnetic spectrum. It is of interest to note that while the eye can perceive only one octave of ether vibration, the air vibration of sound perceptible to the ear is 7 to 8 octaves.

Before any radiation can exert any action it must be absorbed (Grotthus's Law). Thus the visible rays pass through the

transparent media of the eye and are absorbed by the retina, where those chemical molecules which have the same rate of vibration as the absorbed rays are set into resonant vibration reaching an amplitude or intensity great enough to produce chemical change. The photochemical effects thus produced on the rods and cones of the retina are transmitted as light impulses to the brain. The infra-red (heat) rays have great power of penetrating not only the body but the atmosphere. They penetrate the skin and are absorbed by the blood, being thus recognised as heat. The Hertzian waves have no known effect upon the body, and are recognised only when they are transformed by special apparatus into sound waves. The ultra-violet rays have very little penetrating power and are easily stopped by atmospheric impurity. Of these rays, the longer ones pass through the skin to be absorbed by the blood, where they produce certain physiological effects which make them of such great therapeutic value (see p. 486 *et seq*). Many of these solar ultra-violet rays, however, are absorbed before they reach the earth. Clouds, atmospheric pollution such as dust, dirt, smoke and moisture form effective barriers against their passage, whilst ordinary glass (such as windows) also stops a great portion of them. According to Leonard Hill, the smoke pollution of the atmosphere in big cities cuts down the ultra-violet rays by one-half to two-thirds in comparison with clean country and seaside places. This is an additional effective argument for the campaign against smoke. It is probable that with the elimination of the smoke pollution of the atmosphere by the use of gas or smokeless fuel, rickets and pulmonary disease would rapidly disappear. Clothes also act as a screen which shut out some of the ultra-violet rays. According to Hess, thin nainsook cotton will prevent a certain proportion of these rays from reaching the body, and thick nainsook cotton will act as a barrier against a larger proportion of these rays. Artificial silk, which is an acetyl-cellulose product, is according to Hill, more transparent to ultra-violet radiation than natural silk or wool, which are protein products. "What with mist, cloud and smoke, glass windows which cut out rays shorter than about 530-320 $\mu\mu$, and clothes," says Hill, "city people get almost no exposure to ultra-violet rays during winter, and while every effort should be made to prevent smoke pollution by use of smokeless fuel, and to educate people to expose their skin to the sunlight whenever opportunity occurs, a great deal can be done for the general health by means of arc baths. *These should be instituted not only in hospitals, but in schools and in public baths.*"

Recently a new glass, called Vitaglass, has been introduced by F. P. Lamplough. This contains a large proportion of rock-crystal or quartz, which transmits rays up to 275 $\mu\mu$ (2,750 Å), i.e., practically all the therapeutic rays.

In 1926, the Smethwick Education Committee equipped a number of class-rooms in selected schools with Vitaglass, and after a period of nine months compared the heights and weights of the children in these rooms with those in adjoining rooms in the same schools, and found that these gained half an inch more in height and three pounds more in weight than the others. The matter needs further observation and confirmation.

PHYSIOLOGICAL EFFECTS OF ULTRA-VIOLET RAYS.

We have seen that the effects of any radiation only make themselves felt after they are absorbed. The retina, for instance, absorbs all the rays of the visible spectrum, and hence the eye can appreciate all the various colours either singly or in combination. Any substance which fails to absorb any visible radiation of a particular wave-length will fail to show that colour which is due to the particular wave-length of that radiation. Most of the constituents of living cells do not absorb any visible rays and are therefore colourless. They, however, absorb ultra-violet rays in various degrees. All rays shorter than 300 $\mu\mu$ (3,000 Å), i.e., all ultra-violet rays, are absorbed by a layer of epidermis 0·1 mm. thick. The shorter rays of this region are absorbed by the horny surface of the epidermis, and therefore do not affect the deeper-lying living cells. The latter are acted upon by the middle region waves, causing erythema or redness, and, if sufficiently strong, produce "sunburn." Probably no rays shorter than 240 $\mu\mu$ penetrate the horny layer. Rays longer than 330 $\mu\mu$ penetrate and are absorbed by the blood, where they are converted into long heat rays. It is seen, therefore, that whilst the living cells of the epidermis absorb a considerable number of ultra-violet rays, the number of such rays which reach the blood is very small. Any physiological effects of the ultra-violet rays on the body must therefore be indirect, by the excitation of chemical changes in the epidermal living cells, which produce substances that are absorbed into the blood and act also upon the nervous system.

The effects may be summarised as follows :—

(1) Skin.

(*a*) A few hours after exposure the part becomes warm and red. This *erythema* varies in severity with the dose of the radiation, and the susceptibility or sensitiveness of

the patient from a mere feeling of warmth to actual inflammation of the skin.

(*b*) *Pigmentation.*—Following repeated doses of the rays, the erythema is followed by a bronzing of the skin which is due to the deposition of melanin granules, which latter act as a screen and protect the cells from an overdose of the very short rays. It has been shown experimentally that waves shorter than 290 $\mu\mu$ (or 2,900 Å) produce violent erythema, and that these rays cannot pass through the melanin pigment which is produced in the body by rays between 290 and 330 $\mu\mu$ (2,900-3,300 Å). The pigment is of great therapeutic value in another way, for it transforms absorbable rays of short wave-length into penetrating rays of longer wave-length.

(*c*) The cholesterol in the skin possibly becomes activated with the production of the anti-rachitic vitamin D (see pp. 353 and 496).

(2) **Vascular System.**—By melanin converting some of the rays into longer-waved heat rays, dilatation of the cutaneous blood-vessels is produced, resulting in a fall of blood-pressure.

(3) **Blood.**—The effect of the ultra-violet rays on the blood is not a direct one, since, as we have seen, the shorter or most active rays are absorbed by the skin and do not reach the circulation at all. It is probable, therefore, that the effects on the blood are produced by photochemical bodies produced in the skin and absorbed into the blood. These effects are the following:—

(*a*) The number of red cells, the amount of hæmoglobin as well as the lymphocytes, and also the platelets, are increased in number, and hence the value of these rays for anæmic conditions (other than leukæmias, in which the lymphocytes are in such abundance).

(*b*) The clotting power is diminished, thus rendering susceptible patients liable to hæmoptysis.

(*c*) The calcium and phosphorus contents are raised, thus accounting for the beneficial effect of the ultra-violet rays in rickets. The increased concentration of these salts in the blood is probably due to the better absorption from the bowel, owing to the decrease of intestinal bacteria. The following table is given by B. Kramer and F. H. Boone (quoted by Hill), showing how the percentage of these salts was increased in rickety children, from subnormal to normal (viz., Ca about 10 mg. per 100 c.c. and P about 6 mg. per 100 c.c.), as the result of exposure to light:

TABLE CXLVII.

	Calcium	Phosphorus	
10.6.22	8·8 mg. per 100 serum	2·8 mg. per 100 serum	Marked rickets
2.8.22	11·1 ,, ,,	5·0 ,, ,,	Definite healing
8.8.22	11·0 ,, ,,	3·0 ,, ,,	Moderate rickets
24.8.22	11·2 ,, ,,	5·9 ,, ,,	Healed rickets
11.8.22	10·6 ,, ,,	3·0 ,, ,,	Rickets
7.9.22	9·2 ,, ,,	5·9 ,, ,,	Healed rickets
21.8.22	7·9 ,, ,,	3·1 ,, ,,	Rickets
7.9.22	9·0 ,, ,,	5·1 ,, ,,	Healing

Two minutes' daily exposure to the mercury vapour lamp (see p. 490) at a distance of 3 ft., prevents the development of rickets in rats kept in darkness and fed on rickets-producing diet, and similar doses of rays given to a baby will double the mineral contents of its blood in a fortnight.

(*d*) The **bactericidal power** of the blood is increased, and hence the value of these rays in bacterial diseases such as chronic boils, &c. Excessive dosage of light lowers this power.

(4) Owing to the raised temperature of the blood (see p. 486) the **general metabolism** of the body and hence also the appetite is increased. The respiratory exchanges are increased and growth of the body is accelerated. *Nursing mothers get an increased flow of milk to their breasts. Also the milk acquires antirachitic properties.* It has been found at Alton that the metabolic activity of children undergoing natural heliotherapy is, in spite of their recumbent and immobilised condition, 40 per cent. higher than that of an ordinary child. This may, however, be due to the cooling effects of the air.

(5) **Nervous System.**—It has recently been shown that the mental condition of children, as ascertained by intelligence tests (after eliminating possible errors connected with heredity, &c.), is improved in children to the extent of one year by exposure to ultra-violet rays (Macrae and Gauvain).

This is seen from the following table[1] :—

TABLE CXLVIII.

	Alton Hospital (sunlight)	Special schools
Number of cases	62	117
Average age	11·85 years	11·13 years
Average mental age ..	10·17 ,,	9·18 ,,
Average mental ratio ..	90·40 ,,	82·50 ,,

[1] *Medical Annual*, 1926, p. 575.

Heredity, educational opportunities, and the nature of the disease from which the children were suffering were allowed for.

(6) **Eyes.**—The conjunctiva as well as the cornea absorb rays of less than 295 $\mu\mu$, and become inflamed, and hence the eyes must be protected during exposure by means of suitable blue-tinted goggles (see fig. 133). Snow-blindness is due to the effect of ultra-violet rays reflected from it, snow being a good reflector of these rays.

Natural Sunlight.—It is as impossible for a child to thrive without sunshine and fresh air as it is for it to live without food. Exposure of normal babies to direct sunlight should begin when they are a couple of weeks old. In our climate it is generally possible to begin outdoor sunbaths about April or May, so that babies born in spring or summer can have outdoor sunbaths earlier than those born in winter. Indeed, statistics prove that babies born in winter are more likely to develop rickets than those born in the summer, because they are deprived of sunlight during their most rapid-growing period. A spot is selected which is protected from the wind, and the baby is put in the direct sunlight, lying first on one side then on the other, so that each cheek is alternately exposed and yet its eyes are kept away from the direct sunrays. On the first day its hands are exposed for about ten to fifteen minutes until they are slightly reddened. The exposure is daily increased by three to five minutes, until it reaches one hour in the morning and one hour in the afternoon. After a few days the exposed parts become tanned. The surface exposed is also gradually extended, by first rolling up one sleeve, then both, and later the legs and thighs are exposed. In the very hot summer months the sunbaths must be given before 10 a.m. or after 4 p.m. When the sun is not too hot, toddlers should be allowed to run about in the back garden with no clothing except short pants and sandals.

Babies born in the winter should be given indoor sunbaths in front of an open window and protected from draughts. Owing to the deficiency of ultra-violet radiations in the winter the exposures should be longer.

Relation between Natural and Artificial Sunlight.—Whilst in sunny, dry climates, at altitudes above the smoke and dirt, exposure to natural sunlight (except in tropical plains where the air is stagnant and the sun very hot) is in many respects superior to treatment by artificial substitutes (because we get the additional benefits of the fresh air in motion), there is no question about the

superiority of the ultra-violet rays as produced by the artificial lights in climates like England, where the valuable rays are shut out by clouds, smoke pollution, &c. As Rollier says: "Sun and ultra-violet rays bear much the same relation to one another as crude drugs do to their synthetically-prepared chemical substitutes." Dr. Humphris elaborates this remark as follows: "If we wish to give a patient a drug to relieve pain, do we give a handful of poppies, make a decoction of this, and order a mouthful to be taken at intervals, or do we prescribe a hypodermic injection of morphine, an accurate dose of exactly that part of the poppy that is desired? It is this accuracy of dose in actinotherapy which makes for the superiority of artificial sunlight over Nature's best efforts." A well-ventilated treatment room is, however, essential.

Sources of Artificial Ultra-violet Radiation.—There are, for practical purposes, three sources of ultra-violet rays at our disposal:—

(1) The carbon arc lamp. } (open lamps).
(2) The tungsten arc lamp }
(3) The mercury-vapour lamp (closed lamp).

The first two kinds of arc lamps are *open*, i.e., their flame is not enclosed in any way. All arc lamps should be automatic in their regulation, so that the flame is constant, otherwise the flame becomes longer as the carbons burn away, and the intensity of the emitted rays consequently varies during exposure. The mercury-vapour lamp is enclosed, and has a quartz window which is transparent to ultra-violet rays, in contradistinction to glass which is opaque to them.

There are several varieties of each of these lamps, details about which will be found in books specially devoted to the subject (see literature at end of chapter).

The mercury-vapour lamp (which was first devised by Wheatstone in 1835) has the advantage over the open arc lamps in that it consumes a smaller amount of current, and has a higher percentage of ultra-violet and lower percentage of infra-red or heat rays as seen from Table CXLIX), thus very much shortening the necessary exposure. It also burns steadily and silently, unlike the carbon arc which often makes a hissing sound and emits sparks, an important point in the treatment of young children. Its disadvantages are that it is fragile, the tendency for the mercury to become deposited on the quartz window and obstructing the rays, and the tendency in the quartz itself to become less transparent to these rays.

Fig. 132.—The children's sun balcony at Leysin.

The following table gives the percentages of the various radiations given off by various sources. (Pacini, quoted by Russell and Russell) :—

TABLE CXLIX.

Source	Infra-red	Light	Ultra-violet
Mercury vapour	52 per cent.	20 per cent.	28 per cent.
Sunlight	80 ,,	13 ,,	7 ,,
Open arc lamp (carbon)	85 ,,	10 ,,	5 ,,
Incandescent lamp	93 ,,	6 ,,	1 ,,

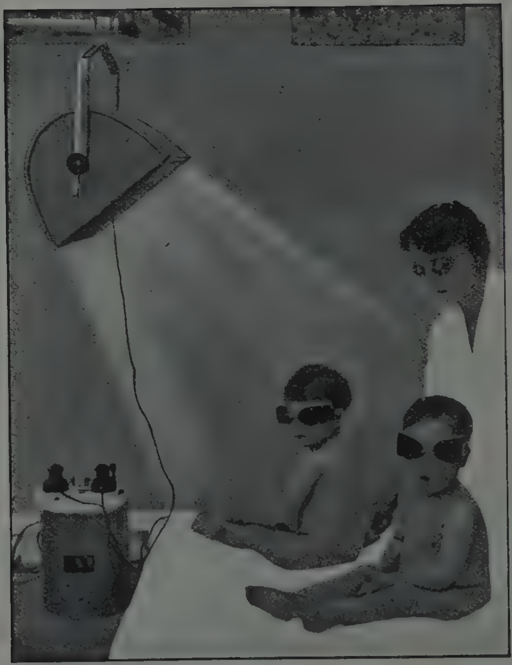


FIG. 133.—Children under ultra-violet ray treatment.

The advantage of all the lamps over natural sunlight, at any rate in climates like our own is, as I have already pointed out (p. 490), that we can regulate the dosage of the rays that the patient is to get with almost mathematical exactitude.

Dosage.—To determine the intensity of the rays several methods may be employed, but the one most commonly and conveniently used is Leonard Hill's acetone-methylene-blue method. A 30 per cent. solution of acetone, containing a measured quantity of a standard solution of methylene blue, is placed in a small quartz tube and exposed to the rays from the lamp, at a given distance for a given time. The amount of acid produced by the action of the rays during that time is estimated by the degree of *bleaching* which is determined by comparison with standard blue tints. One degree signifies two to four times as much ultra-violet radiations as are required to produce erythema of average white skin. In this way it is possible to gauge the decreasing intensity of a burner with time and the correspondingly longer exposure needed. Another apparatus is the "Keller" dosimeter.

The minimal erythema dose in a normal white skin usually corresponds to twice the time required for killing a standard culture of infusoria in a standard quartz cell and at a temperature of 20° C. The infusoria-killing (I.K.) unit is the time required to destroy infusoria placed in a water-cooled quartz cell of standard width at 20° C.

The minimal erythema dose is two I.K. units. A sample of blood taken by venipuncture is placed in a sterile test tube and defibrinated. Two and a half hours after exposure to five I.K. units, another sample of blood is taken and defibrinated, and the bactericidal power is examined and recorded as follows :—

TABLE CL.

	W/6	W/18	W/36	W/60
Number of colonies of staphylococci which were implanted with each 50 c.c. of blood	340	113	57	34
Number of colonies of staphylococci which grew in each 50 c.c. of blood at 2 p.m. (before radiation)	68 (20 %)	22 (20 %)	12 (21 %)	7 (20 %)
Number of colonies of staphylococci which grew in each 50 c.c. of blood at 4·30 p.m. (after radiation)	6 (2 %)	2 (2 %)	1 (2 %)	0 —

The average hæmobactericidal power of the blood before radiation = 80 per cent.; average hæmobactericidal power after radiation = 98 per cent. This method enables one also to control the treatment of feeble patients who have low hæmobactericidal powers.

Factors on which the Reaction of the Skin to Ultra-violet Radiation depends.—(1) The intensity of the source of the ultra-violet radiation; (2) the distance from the source of light—in accordance with the law of inverse squares; (3) the temperature; (4) the individual sensitiveness of the skin; (5) the angle of incidence of the rays, the maximum effect being produced by rays falling perpendicularly on the surface. Hence with oblique rays a longer exposure is needed. For details see books on heliotherapy.

Indications for Heliotherapy in Children.—Whilst no extravagant claims should be made for heliotherapy as a panacea for all

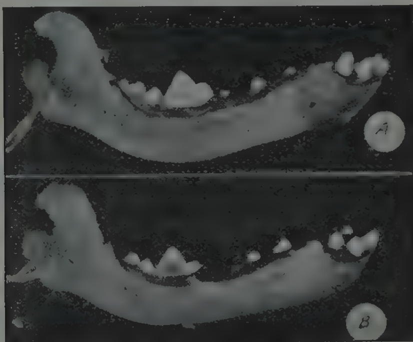


FIG. 134.—The effect of ultra-violet radiations. Photographs of lower jaw of two puppies brought up on the same diet deficient in calcifying vitamin and living under the same conditions. Puppy A only was exposed thrice weekly for twenty minutes to the radiations of a mercury-vapour lamp. Note the better formed teeth of A as compared with B. (Mellanby, *Brit. Med. Journ.*, March 20, 1926.)

diseases and conditions of childhood, there is no doubt of its great value in the following conditions:—

(1) **Rickets.**—This can be definitely and absolutely prevented and cured, as shown by the clinical disappearance of the various symptoms, by the X-ray appearances of the bones, and by the increase in the mineral concentration of the blood. Teeth which had been delayed also appear (fig. 134). Huldschinsky was the first, in 1919, to show the curability of rickets by ultra-violet rays. In children up to about $2\frac{1}{2}$ years rickety deformities of bone become straightened out.

Herodotus, as stated on p. 20, attributed the hardness of the bones of the heads of Egyptians, as well as their lack of baldness, to their custom of shaving their heads from early youth and exposing them to the sun. The Persians, on the contrary, had soft skulls "in consequence of their keeping the head covered from the sun." Indeed, on the field of battle where the Egyptians and the army of Cambyses fought, it was pointed out to him, says the historian, that the skulls of the Persians were so soft "that you could perforate them with a small pebble," while those of the Egyptians were so hard "that with difficulty you could break them with a large stone."

(2) **Surgical tuberculosis** (tuberculous bones, joints, peritoneum), as well as root tuberculosis (i.e., tuberculosis of the glands at the root of the lungs) are benefited. Tuberculous bones and joints are cured, but in the case of the other conditions the results, though favourable, are not quite so good. Tuberculous peritonitis may take a couple of years to cure.

A. Eidinow[1] has shown that tubercle bacilli lose their virulence (as tested by inoculation of guinea-pigs) after 10 minutes' exposure to rays ranging between 7,620 and 2,300 Å, or after 30 minutes' exposure to rays ranging between 5,720 and 2,800 Å, but that they retain their virulence even after an hour's exposure to rays of wave-lengths 5,720-3,300 Å. Hence it is only the short rays 3,300-2,300 Å that have a direct bactericidal action, and the shorter the rays the greater their action. As, however, these short rays do not penetrate the deeper layers of the skin it appears that any good effects of ultra-violet rays upon deep-seated tuberculosis lesions can only be an indirect result brought about by the long heat rays producing tissue reactions (see p. 486).

(3) **Chronic eczema** is improved, and so is psoriasis, &c.; chronic furunculosis is cured.

(4) **Malnutrition and wasting**, as well as pretuberculous conditions in infants and children, are greatly benefited, as are also those recovering from acute infections like measles. Babies that are not gaining are found by Dr. Dora C. Colebrook, of the North Islington Welfare Centre, to begin to gain on exposure to the rays.

(5) The various **simple anæmias** of childhood.

(6) **Nervous and irritable infants and children** are said to become peaceable, happy and contented.

(7) **Nursing mothers whose lactation is failing** improve markedly. Out of sixteen mothers twelve reacted well after eight exposures.

[1] *Brit. Med. Journ.*, July 30, 1927.

(8) **Irradiation of anæmic women during pregnancy** would probably benefit the fœtus.

Susceptibility of Different People to Ultra-violet Rays.—Different people respond in different degrees to the action of these rays. Thus, people with dark complexion tolerate higher doses than fair-complexioned ones. Children require half the adult dose and infants need only half that of children. Females require three-quarters the dose of males, and parts of the body continually exposed to the sun (e.g. face and hands), tolerate larger doses than parts normally covered, such as the abdomen. Certain pigments also affect the susceptibility in one or other direction. We have already referred to *melanin* which *acts as protector*. *Eosin*, or *hæmatoporphyrin* (the iron-free derivative of hæmoglobin), when injected subcutaneously *renders the body most sensitive* to these rays. This explains the association between hæmatoporphyrinuria (a condition in which hæmatoporphyrin is found in the urine) and the vacciniform eruption known as *hydroa æstivale*, which sometimes occurs as the result of exposure to sunlight.

To test the sensitivity of a person to a first exposure, a cuff is put round the arm, and an area of skin is exposed through a hole in the cuff for five minutes, and another for ten minutes, and the resulting erythema is noted the next day. A dose should be given which produces only the slightest erythema at first.

The Contra-indications to Heliotherapy are: (1) Febrile conditions, because of the heating effects and fall of blood-pressure directly or indirectly produced (see p. 487); (2) albinism, a condition in which the natural pigment is absent; (3) cases of very active metabolism, e.g., exophthalmic goitre.

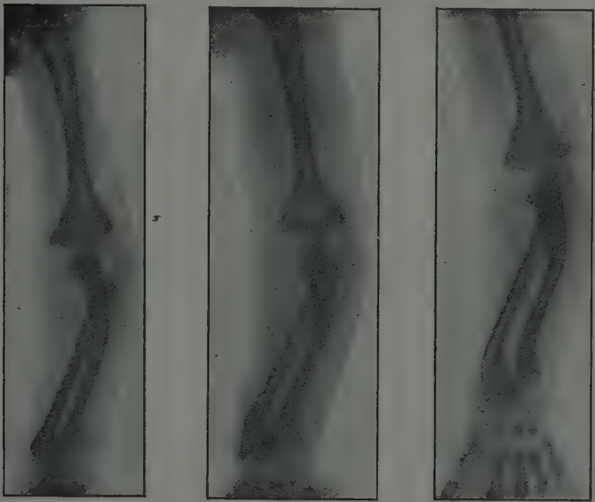
These conditions are not absolute contra-indications, but special care and skill are required in dealing with them.

Unsuitable cases exposed to irradiation may experience a feeling of depression, because of the fall of the bactericidal power of the blood to below normal, after the initial rise. The rate of sedimentation of the red blood-corpuscles is also increased, which means that the patient's resistance is diminished.

Relation between Sunlight and Vitamins.—It has been shown that certain food substances such as wheat, biscuits, flour, meat, eggs, milk and various oils, including vegetable oils, when exposed to emanations from a mercury-vapour lamp, become endowed with antirachitic properties equal to cod-liver oil. Oils

so irradiated resemble cod-liver oil so much in colour and odour that the term "jecorization" has been used for this process. Cholesterol is another substance affected in the same way by ultra-violet rays (fig. 135, *a*, *b*, *c*), and it is possible that one of the factors in the antirachitic effects of these rays on the body is this action of the rays on the cholesterol which is abundant in the skin.

Harriette Chick and Margaret Honoria Roscoe[1] have shown



(*a*) (*b*) (*c*)

Fig. 135.—Effect of irradiated cholesterol in rickets (L. G. Parsons, *Brit. Med. Journ.*, March 20, 1926); (*a*) Radiogram before treatment showing very marked rachitic changes; (*b*) Result of treatment with irradiated cholesterol for three weeks. A considerable degree of healing has occurred; (*c*) Later stage. Bones have completely healed.

that (1) the vitamin A (growth-promoting and antixerophthalmic) content of milk is independent of the diet of the cow, or of the degree of its exposure to sunlight; (2) the antirachitic value of milk (vitamin D content), on the other hand, depends principally upon the amount of exposure the cow has to sunlight; (3) hence milch cows should not be confined in stalls but should be allowed to graze in the open.

[1] *Bioch. Journ.*, 1926, xx, pp. 622-649.

Various kinds of Glass used in Ultra-Violet Radiation Work.—(1) Ordinary glass transmits visible rays but is opaque to ultra-violet rays less than 3,300 Å; (2) Vitaglass transmits both visible and ultra-violet rays (down to 2,800 Å); (3) Chance's ultra-violet glass transmits ultra-violet rays, but is opaque to visible rays; (4) Uranium glass fluoresces under the influence of ultra-violet rays. Hence by allowing ordinary light to pass through Chance's ultra-violet glass, the ultra-violet rays can be made visible by means of uranium glass. In addition, Iceland spar transmits rays down to 2,150 Å, quartz down to 1,850 Å, and fluorite down to 1,250 Å.

Violet and Ultra-violet Rays.—Violet rays is the name popularly assigned to the electrical discharge produced in vacuum glass tubes from small monopolar high-frequency sets often used by hairdressers, beauty specialists and other quacks. It is important that the reader should not confuse these quack apparatus with the real thing. These radiations are entirely free from ultra-violet rays, and cannot therefore produce the same effects.

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CHAPTER XXI

THE PHYSIOLOGY AND HYGIENE OF THE PREMATURE AND CONGENITALLY DEBILITATED INFANT

"He (the premature infant) is like some dweller in the hot plains of India who has been transported in a moment of time on some 'Magic Carpet of Tangu' to the 'chill summits of the frosty Caucasus,' with no opportunity for acclimatising such as a gradual transit affords."—BALLANTYNE.

Definition.—Every infant that is born before full term, i.e., before the end of the 40th week or 280th day, is, of course, premature, but when we speak of a *premature* labour we mean the birth of a premature *viable* infant, viz., in general after the 28th week of pregnancy. The term *congenital debility* applies to those weakly infants that have been born at or near full term, but which for some reasons connected with their ante-natal or intra-uterine nutrition are not so big or strong as normal infants born at full term. For instance, a case has been recorded of an infant born at 272 days, i.e., near full term, whose birth weight was only 500 grm., or 1 lb. 2 oz. Such an infant from the point of view of fœtal age is practically mature, but regarded from the standpoint of function or vitality it is necessarily premature, or, at any rate, immature. Hence, from the point of view of this chapter, it would be best to consider the term "prematurity" or "congenital debility" as practically synonymous and signifying *fœtal malnutrition.*

Diagnosis of Prematurity.—There are many facts which go to show that a baby is premature, but the chief are its weight and length. It is generally agreed that an infant is premature if its weight is less than 2,500 grm. ($5\frac{1}{2}$ lb.), and its length is less than 45 cm. (18 in.). Such an infant requires special care and attention. The normal birth weight and length are, of course, 7 lb. and 20 in. respectively. The other signs of prematurity, such as the relative sizes of the circumferences of the head and shoulders, the characters of the skin and nails, the characters of the blood,

feebleness of the cry, the degree of ossification of the various bones, as shown by X-ray examination, &c., are of scientific and medico-legal interest, but do not concern us here. It may, however, be noted that even X-ray findings cannot be taken as giving absolutely reliable data for determining fœtal age with mathematical certainty. Thus, the centre of ossification of the lower epiphysis of the femur, which is usually taken as a sign of maturity, has been found in a premature baby weighing less than 2 kg. (Ylppö).

Incidence and Mortality.

(*a*) *Incidence.*—There are no official statistics, but it is probable that 10 per cent. of all live births is about the right percentage of prematurely born infants. This corresponds to about 74,000 premature births in this country in 1923. In addition there are, of course, a very considerable number of premature stillbirths.

In the Moscow Foundling Hospital, between 1869 and 1880, there were 5 per cent. of children whose birth weights and lengths were less than 2·5 kg. and 45 cm. respectively. Budin[1] found 658 out of a total of 6,151 births in the Clinique Tarnier (1898-1901) to have been congenitally debilitated (= 10·7 per cent.), and Pinard,[2] out of 188,204 infants born in the Paris Maternité and Clinique Baudelocque, found 29,071 (i.e., 15·4 per cent.) to have had a birth weight of less than 2·5 kg.; Brandt,[3] out of 5,439 births at a maternity home between 1913 and 1922, found 566 (= 10·4 per cent.) to have weighed less than 2·5 kg., as follows:—

Under 500 grm. birth weight		12 =	0·22	per cent.
500-1,000 ,,	,,	45 =	0·83	,,
1,001-1,500 ,,	,,	65 =	1·21	,,
1,501-2,000 ,,	,,	129 =	2·37	,,
2,001-2,500 ,,	,,	315 =	5·79	,,
Total premature		566 =	10·42	,,

Very similar figures are given by Schödel.[4]

(*b*) *Mortality.*

(*a*) *As a cause of infant mortality*, prematurity was responsible for 23 per 1,000 live births in 1923, which is exactly one-third of the total infant mortality for that year (viz. 69 per 1,000). Hence, if we could prevent the birth of premature infants, or preserve

[1] *La Presse Médicale*, 1902, No. 97.

[2] Quoted by C. Hahn, "Des prématurés, caractères, pronostie, traitement." *Thèse de Paris*, 1901.

[3] *Monatschr. f. Kindesheilk.*, 27, 1923, 210.

[4] *Münch. med. Wochenschr.*, 1923, 1,175.

their lives after birth, we could at once reduce the infant mortality to about two-thirds its present rate, i.e., to 45 to 50 per 1,000.

(β) *As a cause of neo-natal mortality*, prematurity was, in 1923, responsible for 19 per 1,000, i.e., 60 per cent. of the total neo-natal mortality (32 per 1,000). Hence, we see that (i) the great bulk of prematures, i.e., 19 out of 23, or over 80 per cent., died during the first month of life; (ii) the problem of further reducing infant mortality is mainly a problem of preventing the incidence and mortality of premature infants; (iii) **the great problem of neo-natal mortality is the problem of prematurity.**

(γ) *Fatality-rate amongst Prematures.*—Statistics show that 50 per cent. of all premature infants die during the first year of life. We have just seen that the infant mortality amongst normal babies (i.e., after excluding all prematures) is about 45 to 50 per 1,000. Hence we see that **premature babies die ten times as rapidly as normal ones.**

(δ) Statistics also show that the longer a premature infant has survived after birth the greater is its chance of survival, and if it has reached the age of one year its chances are practically the same as those of any other infant (see Table CLX).

Causes of High Mortality of Premature Infants.—The chief troubles with a premature baby are that its tissues are too delicate to withstand the injuries to which it is subjected during its passage through the pelvic canal, so that the incidence of stillbirths, as well as of very early neo-natal deaths, is very high among them; and on account of its peculiar anatomical and physiological characters it is not properly fitted to the external post-natal environmental conditions. For, from the point of view of function, it is still a fœtus requiring intra-uterine surroundings.

Important Anatomical and Physiological Peculiarities of the Premature Infant.

(1) *Proportions of Body.*—The ratio $\dfrac{\text{head circumference}}{\text{shoulder circumference}}$ is less in the premature than in the mature infant, as the following table [1] shows:—

[1] A. Ylppö, "Path.-anat. Studien bei Frühgeburten," *Zeitschr. f. Kinderheilk.*, xx, 1919.

TABLE CLI.

Weight of infant in grm.	Head circumference (cm.)	Chest circumference (cm.)	Ratio between chest circumference and head circumference
Premature—			
Under 1,000 ..	25 0	20·8	0 83
1,001-1,500 ..	31·8	24·5	0·77
1,501-2,000 ..	30·0	26·3	0·88
2,001-2,500 ..	32·3	29·5	0·91
Mature—			
3,000-3,500 ..	33·5	31·0	0·93

The greater the prematurity the smaller is the ratio. The importance of this ratio is from the point of view of intra-natal effects. During birth the relatively larger head receives more compression, and owing to the great delicacy of the intracranial vessels, cerebral hæmorrhages are very prone to occur. Also the fact that the chest circumference is relatively less in proportion to the size of the head than is the case at full term, means that the chest receives relatively less compression in the premature than in the mature infant. Now the elastic rebound of the chest after intra-natal compression helps the lungs to expand and fill with air, and hence the premature infant has a less chance for pulmonary inflation and ventilation than has the mature one. The head is comparatively larger in the premature than in the mature baby and the fontanelles are very big; the lower limbs are also comparatively shorter.

(2) *Thermal Instability.*—We have already learned that new-born infants cannot easily maintain their body temperature on account of the imperfect development of their heat-regulating mechanism. This is so in an excessive degree in the premature infant, for the following reasons.

(*a*) The surface area of the skin is proportionately greater and therefore there is a greater heat loss. Thus a premature infant, weighing say 1,300 grm., and measuring say 40 cm., would have a total surface of 1,123 sq. cm. (by the Du Bois formula), or a surface of about 936 sq. cm. per kilogramme; whilst a full-term infant of 3,500 grm. and 50 cm. would, by the same formula, have a total surface of 2,200 sq. cm., or one of about 621 sq. cm. per kilogramme. In other words, the premature infant has a surface area

$1\frac{1}{2}$ as large per kilogramme body weight as has the full-term baby.

(*b*) Absence of subcutaneous fat affords greater facility for loss of heat.

(*c*) Insufficient oxygen combustion, because:—

(i) The general debility of the infant prevents it from taking in sufficient nourishment to produce heat.

(ii) The collapsed condition of the lungs, due to deficient development of elastic tissue and the smaller size of the alveoli, as well as the defective excitability of the respiratory centre, makes the metabolic processes rather slow and feeble.

(iii) The heat centre in the brain is feebly developed.

In virtue of this thermolabile state, the premature infant loses more heat and its body temperature is lower than that of a full-term infant. Hence it is most important to preserve its body heat in every possible way.

(3) *The circulation* is in respect of certain anatomical structures still of the fœtal type, and owing to the collapsed condition of the lungs, the pulmonary circulation is impeded, so that the ductus arteriosus and foramen ovale tend to remain patent. Moreover, the lumen of the ductus arteriosus gradually becomes narrower in the fœtus during the later months of pregnancy, to prepare it for its ultimate obliteration during the neo-natal period. This narrowing of the calibre of the duct also restricts the amount of arterial blood flowing to the respiratory centre, thus rendering the latter more excitable and ready to assume independent activity when the infant is assailed by extra-uterine stimuli. Hence, in a prematurely born infant the respiratory centre is in a torpid state. Also the *blood-vessels* are very delicate and fragile, and hence are liable to traumatic bleeding, unless handled with the greatest gentleness. The premature infant's *blood* also contains a deficiency of immune bodies, and is more acid (as measured by its hydrogen-ion concentration), two facts which predispose it to bacterial infection.

(4) The amount of mineral salts (calcium and iron, &c.) retained by a fœtus during the last three months of pregnancy is, as we have seen, greater than that retained during the whole of the first six months, and hence premature babies are especially liable to suffer from rickets and anæmia.

For all these reasons the premature expulsion of a fœtus is a

calamity the importance of which cannot be exaggerated. "The premature infant," says Ballantyne, "is partly a fœtus and partly a new-born child. In him is seen fœtal physiology endeavouring to cope with neo-natal surroundings, but in him also is seen neo-natal physiology hampered in its action by persistent fœtal conditions."

Factors which Influence the Mortality of Prematures.

The chances of a premature infant to live depend upon various circumstances, which may be classified as :—

(a) *Ante-natal influences.*
(b) *Intra-natal influences.*
(c) *Post-natal influences.*
(d) *Ætiological influences.*

(a) *Ante-natal Influences.*

(i) **Fœtal Age.**—The younger the fœtus the less are its chances of survival after birth (see Table CLXII). It must, however, be remembered that it is extremely difficult to determine the exact age of a fœtus, since the beginning of pregnancy cannot be determined with any certainty. The only method which is at all accurate is the determination of the degree of ossification of the various bones by means of the X-rays. There is a fairly definite fœtal age at which the centres of ossification of the various bones appear, and these centres can be recognised on the X-ray plate.

(ii) **The physiological development,** as determined by :—

(α) *Weight.*—The smaller the birth weight the less are the infant's chances of survival. Thus, from Table CLXI we see that the chances of an infant weighing more than 2 kg. ($4\frac{1}{2}$ lb.) are about eleven times as great as those of an infant weighing less than 1 kg. ($2\frac{1}{4}$ lb.). The smallest premature infant that I have personally known to survive, and is now 5 years old, had a birth weight of $1\frac{3}{4}$ lb. (or about 800 grm.), but a case has been recorded of an infant weighing only 500 grm. (i.e., just over 1 lb.) that has been saved.

(β) *Length.*—The shorter the infant the less are its chances of survival. The shortest infant that has been saved measured 31 cm., i.e., about

12 in. Its birth weight was 750 grm. (i.e., 1 lb. 10 oz.).

(γ) *Body Temperature.*—The lower the temperature the greater the mortality. The body temperature, as we shall see later, depends, amongst other things, upon the degree of development of the central nervous system (see Table CLXI).

(δ) *The development of its feeding apparatus*, as represented by the nervous mechanism concerned with swallowing, and the state of its sucking muscles. The feebler the development of the feeding mechanism, the less are the chances of survival.

(ϵ) *The Development of its Respiratory Apparatus.*—A baby that can cry generally survives, whilst one that is very somnolent has its chances of survival considerably reduced.

(iii) **The Presence or Absence of Abnormalities.**—It is clear, for instance, that a premature baby with a cleft palate would be very difficult to feed and keep alive.

(iv) **The Presence or Absence of Transmissible Disease, or Predisposition to Disease.**—Thus, the premature babies of tuberculous or untreated syphilitic mothers have a very poor chance.

(*b*) *Intra-natal Influences.*—Any obstetrical manipulations on a premature infant will jeopardise its life.

(*c*) *Post-natal Influences.*—Under this heading come the general care of the baby, and the skill with which that care is given. We shall deal with this subject later (see p. 509 *et seq.*).

(*d*) *Ætiological Influences.*—By this is meant the causes which bring about prematurity. The causes of prematurity are similar to those of fœtal death, with which I have dealt fully in Chapter IV (p. 98).

The prospect of congenitally syphilitic babies is worse than those of tuberculous mothers in the proportion of 2 : 1 (mortality of syphilitics = 73 per cent., and that of tuberculous = 33 per cent.), but the mortality of the tuberculous is worse than that of other prematures. Eclamptic or albuminuric prematures have a high mortality during the first day, because of the intra-natal injuries resulting from the rapidity with which the infants are born either spontaneously or by induction. When premature labour is

artificially induced, the infant mortality depends upon the cause; in the case of contracted pelvis in an otherwise healthy mother, the baby's prospects are much brighter than in a case where induction is done to relieve the mother of the strain, such as albuminuria, &c.

The Preventable Causes of Prematurity.

Although the list of causes of prematurity is long, those which are definitely avoidable are not very many, and may be classified into:—

(*a*) *Those due to toxæmia of pregnancy*, e.g., albuminuria, eclampsia, &c. (For prevention, see p. 279.)

(*b*) *Those due to circulation of poisons in the mother's blood*, e.g., syphilis, alcohol, lead, &c. (see Chapters IV and VIII).

(*c*) *Those due to injury.*

(*d*) *Overwork of the mother*, as a cause of prematurity, ought to be prevented in the case of the poorer mothers by the provision of domestic helps to do the more strenuous housework, such as scrubbing, washing, &c., in the last weeks of pregnancy. Under ideal conditions one would have wards in hospitals where mothers can spend their last weeks of pregnancy under proper medical and nursing supervision.

(*e*) As regards *induced premature labour*, the possible method of prevention is in suitable cases such as pelvic obstruction, &c., to wait till full term and do Cæsarean section. In other cases induction should be put off for as long as possible, compatibly with the mother's safety.

Ante-natal supervision will be instrumental in reducing the incidence of prematurity to a certain extent (see Table CLVIII), but by no means entirely, since statistics show that the unavoidable and unknown causes are responsible for some 75 per cent. of all cases of premature birth. The infant mortality amongst prematures is, as we have seen, 50 per cent., but this high mortality can be very greatly reduced by proper skilful neo-natal and subsequent care.

The Physical and Mental Development of Premature Infants.

We have seen that about 40 to 45 per cent. of premature children survive to reach the fifth year of life. Until the survivors have reached that age they are smaller in height as well as in weight than children of the same age that had been maturely born. But after the age of 5 years the average weight and height of both

classes of children are about the same, and some have lived to over 80. The mental development of the premature infant is also retarded at first. This is only to be expected in view of the fact that the cerebral sulci and convolutions in the premature infant are not yet fully developed. It begins to smile at 4 to 6 months, and to walk and talk at a later period than the mature baby, the period of delay varying with the degree of prematurity. Stammering is not an uncommon, though only transitory, defect of speech amongst very premature babies. But such master minds as Newton, Rousseau, Voltaire, D'Alembert, Victor Hugo, Cuvier, Lamartine and Renan were all premature babies.

Ylppö found amongst the 668 premature cases of the Kaiserin Auguste-Viktoria House, in Berlin, 7·4 per cent. idiots and imbeciles, apart from children that were mentally backward. Cases of Little's disease of various degrees of severity are also common amongst them, owing to the frequency of intracranial hæmorrhage in the region of the motor centres of the lower limbs on each side.

Rickets is common amongst premature children. This is due to the fact that such infants are not able to digest milk (even its own mother's milk) well. They excrete a great deal of calcium in the form of calcium soaps, thus depriving the skeleton of a good deal of this mineral. Moreover such infants, as we have seen, have been deprived of a lot of calcium that should have reached them through the placental circulation. To prevent rickets in these children, one may give them the syrup of the phosphate of calcium or, better still, daily exposures of artificial sunlight, starting with two minutes' exposure at a distance of 90 cm., and gradually increasing the dose and diminishing the distance until an exposure of thirty minutes at 75 cm. is given. For similar reasons anæmia is very common amongst such children. This is also greatly benefited by ultra-violet irradiation, as well as by the administration of phosphate of iron.

TABLE CLII.

Weight of Infant	Number of cases	Number with hernia	Per cent. with hernia
1,000 grm.	1	1	—
1,001-1,500 ,,	50	42	84
1,501-2,000 ,,	81	31	38·3
2,001-2,500 ,,	73	15	20·5
Total ..	205	89	43·4

Herniæ.—The premature infant is particularly liable to herniæ or ruptures (inguinal or umbilical), as table CLII shows (Ylppö).

These herniæ rarely require operation or cause trouble. They nearly all disappear when the musculature of the abdomen becomes stronger. They must be prevented from coming out by suitable trusses.

The Metabolism of the Premature Infant.

(1) *Chemical Composition of Prematures.*—Langstein and Edelstein[1] analysed two premature infants born at the end of the sixth and the beginning of the seventh month respectively, and who died after one week and at two days respectively. The following table represents in round numbers the average percentage composition of the whole infant. (The figures in brackets represent the percentage composition of the fat, nitrogenous and non-nitrogenous substance of the dry material of the infant. The composition of full-term infants (Camerer and Soldner[2]) is given for comparison):—

TABLE CLIII.

	Solid	Water	Fat	Nitrogenous material	Non-nitrogenous material	Ash	Nitrogen
Premature ..	20	80	5 (25)	11 (55)	1·5 (7·5)	2·5 (12·5)	1·70
Full term ..	28	72	12 (44)	12 (42)	1·3 (5)	2 7 (9·4)	—

From the table it is seen that the premature infant contains more water and less fat than the full-term infant. The chemical composition of the ash also shows that whilst in the full-term infant the sodium potassium ratio is 1 : 1, it is approximately 1 : 1·5 in the premature. The premature baby also contains more chlorine.

The young fœtus contains about 0·25 per cent. of chlorine, i.e., practically the same as the blood. As the child grows the percentage of chlorine falls until it reaches the figure 0·12 per cent. in the adult.

(2) *Retention of Material.*—Rubner and Langstein[3] studied the retention of material in two premature babies of 1,640 and 2,050 grm. respectively. During a period of eleven days, when the average daily gain was 39 grm., one infant received 1·04 grm. of nitrogen per day in the milk and retained 50 per cent., i.e., 0·52 grm., corresponding to $0·5 \times 6·25 = 3·13$ grm. of protein. This is about half the amount of protein that a fœtus of the same age would deposit. The infant utilised only about 64 to 74 per cent. of the total calories contained in the food, the chief loss of calories being in the fat, of which only 42 per cent. was absorbed. The total calories absorbed from

[1] *Zeitschr. f. Kinderheilk.*, xv, 1916.
[2] *Zeitschr. f. Biol.*, xxxi, 1900; xl, 1900; xliii, 1902; xliv, 1903.
[3] *Arch. f. Physiol.*, 1915, p. 39.

the food were 126 per kilogramme per day, of which 73 were used for heat production and 53 were deposited in the growing infant.

Langstein and Edelstein give the following balance sheet for a premature baby's metabolism :—

TABLE CLIV.

Intake	Retention		Heat production	
	Total	Per cent. of intake	Per kilo	Per square metre
125 grm.	53	42	72	973

The 42 per cent. retained was represented by :—

Protein	3·38	per cent.
Fat	14·61	,,
Water	31·7	,,
	49·7	,,

In other words. for every 100 grm. addition in weight there was deposited :—

Protein	6·8 grm.
Fat	29·0 ,,
Water	64·0 ,,

They believe that 83 calories per kilogramme to be approximately the maintenance ration of a premature baby.

(3) *Retention of Salts.*—Hamilton[1] and Lichtenstein[2] have also studied the metabolism of salts in prematures, especially calcium, phosphorus and iron. Hamilton finds that during the first couple of months the amount of calcium deposited was less than in the case of normal babies, thus giving, as we have seen, an additional reason for the development of rickets in premature babies. In respect of phosphorus, the premature does not differ from the full-term infant. With regard to iron, Lichtenstein finds that premature infants lose more iron than they ingest. They lose at the rate of 0·25 mgm. of iron per day.

(4) The *basal metabolism* of premature infants has been independently studied by Talbot and his co-workers,[3] as well as by Murlin and Marsh.[4] They found the basal metabolism to be very low and quite against the accepted law of surface area, since per unit of body weight it is less in these babies than in mature ones, when the contrary should be the case, since smaller babies have a greater surface per unit of weight.

Intra-natal and Post-natal Care of the Premature Infant.— The chief troubles with a premature baby are that its tissues are

[1] *Am. Journ. Dis. Ch.*, 1920, xx, 316, and *Acta Pædiatrica*, 1922, ii, 1.

[2] *Acta Pædiatrica*, 1921, i, 194.

[3] *Proc. Soc. Exp. Biol. and Med.*, 1922, xix; *Am. Journ. Dis. Ch.*, 1922, xxiv; and *Monatsschr. f. Kindesheilk.*, 1924, xxvii, 470.

[4] *Proc. Soc. Exp. Biol. and Med.* 1922, xix.

too delicate to withstand the injuries to which it is subjected during its passage through the pelvic canal in the process of birth so that the incidence of stillbirths is very high amongst them and that on account of its peculiar physiological characters it is not yet properly fitted to the external post-natal environmental conditions. For, from the point of view of function, it is still a fœtus requiring intra-uterine surroundings. It cannot, for reasons given above (p. 502), stand loss of heat. Further, it cannot take food easily, because its sucking muscles as well as nervous mechanism are very feeble, and its digestive system not yet mature. Moreover, such an infant is peculiarly susceptible to septic absorption, because (1) it lacks immune bodies coming from its mother; (2) its blood acidity is higher; (3) its leucocyte-forming organs are not properly developed. Hence, in addition to the special obstetrical skill that a premature fœtus needs at the hands of the accoucheur, the special post-natal care that such a baby requires consists of:—

(1) **Preservation of body heat**, by keeping it warmly wrapped up in cotton-wool, near a fire, with hot water-bottles or electric heaters in the cot. It must not be bathed, but it can be kept clean by rubbing it with warm olive oil, which not only has a cleansing action but helps to keep in its heat by retarding evaporation (fig. 79, p. 312), and also possibly provides it with some nourishment by its being absorbed through the skin. (For the use of incubators, see p. 516.)

(2) **Proper Feeding.**—The best food is its mother's milk. If it cannot suck, as is the case with very feeble babies, or with primiparæ in whom the flow of milk is very scanty, the milk should be withdrawn from the breast under proper aseptic precautions, and given to baby by means of a spoon or pipette, or, if its swallowing reflex is feeble, and the milk dropped into the mouth flows out again, by means of a stomach tube, consisting of a sterilized No. 10-12 Nelaton's catheter passed through the mouth (the nose is too narrow for the purpose) into the stomach. This can only be done by a trained nurse. The pharyngeal reflex being absent in very premature babies, the passing of the catheter does not produce any retching. Indeed, the whole process, including the introduction of the requisite quantity of milk, can often be done without waking them. If the mother does not secrete sufficient milk, the flow may be stimulated by putting a vigorous child to the breast; 100 to 250 c.c. a day (i.e., 3½ to 8 oz.), according to weight of infant, are sufficient during the first few days.

It is generally recognised that during the first twenty-four hours the infant does not require any food at all. Delestre[1] found the following average figures for the quantities of breast milk taken by premature infants (140 in number) :—

TABLE CLV.

Weight of infant	Quantity of milk taken								
	2nd day	3rd day	4th day	5th day	6th day	7th day	8th day	9th day	10th day
	Grm.	Grm.	Grm.	Grm.	Grm.	Grm.	Grm.	Grm.	Grm.
Under 1,500 grm.	125	135	160	165	180	185	215	235	250
1,500-2,500 ,,	190	230	290	310	320	325	330	340	345

One litre (1,000 c.c.), or approximately 1,000 grm. of milk, is equivalent to about 35 oz. of milk.

According to most authors, *the necessary daily amount of milk is one-fifth of the baby's body weight.* This corresponds to a caloric requirement of 120 to 140 calories per kilogramme, or 55 to 63 per lb., so that a baby weighing 3 lb. would require approximately 180 calories. This amounts to about 9 oz. of milk, to be given at the rate of 1 oz. a feed. If a wet nurse is engaged, her nipples must be well developed to enable the baby to suck. The nurse's baby may be put to the breast of the premature infant's mother in order to stimulate the flow of milk. Another plan is for the mother to put the nurse's baby to one breast at the same time as she puts her own baby to the other.

Intervals of Feeding.—The above table (CLV) represents the approximate amounts of human milk that should be given to babies of different weights during the first ten days of life. They are to be divided into seven three-hourly feeds, but if the baby cannot take so much at one feed, the quantity should be divided into nine or ten two-hourly feeds. The baby should be awakened for its feeds, since the sensation of hunger is not yet developed to wake it, and moreover, the coincident jaundice to which such an infant is particularly liable suppresses any such sensation as the baby may possess.

If the milk is withdrawn from the breast, it must be done with all the necessary aseptic precautions, and should, by preference, be boiled. In the case of babies fed on wet nurses' milk, it is preferable to dilute the milk with equal parts

[1] "Étude sur les infections prématurés," *Thése de Paris*, 1901.

of water, since the baby's digestive organs may not yet be equal to the strain of digesting full human milk.

If breast milk is unobtainable, the baby should be fed on mixtures in equal parts of boiled cow's milk, whey and water, and in case of very feeble babies it may be advisable to peptonize the mixture.

It must be remembered that:—

(1) Breast-fed premature babies begin to gain after the first week, whilst artificially-fed ones may not begin to gain for two or three weeks.

(2) Very feeble infants cannot take more than 100 calories per kilogramme (45 calories per lb.), and sometimes 75 calories per kilogramme (38 calories per lb.) are sufficient.

In other words, very feeble babies may not be able to take more than half the quantity of milk indicated above. As, however, on account of the great warmth under which such babies are kept, they necessarily lose a lot of water by perspiration, the loss must be made good by giving them drinks of water. They must have about one-fifth of their weight of water per day, inclusive of that contained in the milk.

(3) **Prevention of Infection.**—Examination of the blood in prematures gave the following results:—

TABLE CLVI.

Age	Number of cases	Bacteria found in
0-1 days	8	0 = 0 per cent.
2-3 ,,	7	2 = 28 ,,
4-15 ,,	14	10 = 70 ,,

Infection may take place: (1) Ante-natally, through the placenta or liquor amnii; (2) Intra-natally, from the vagina, e.g., ophthalmia neonatorum, or infection of skin wounds, &c.; (3) Post-natally, from the umbilicus skin, gastro-intestinal tract and lungs. According to Ylppö, the gastro-intestinal route is the most important. The ante-natal infection can be considerably controlled by proper ante-natal supervision, and that occurring post-natally may, of course, be prevented by rigid asepsis. The handling of the infant should be done by people who have not been exposed to infection, and with hands suitably cleaned. The umbilical wound should be kept rigidly aseptic and everything used for the infant, such as feeding utensils, &c.,

should be thoroughly sterilised, and hence they should not be kept in the general ward of a hospital. Visitors should, as far as possible, be excluded. If the mother has a cough or a cold in the head, she should wear a gauze mask over her mouth and nose whilst attending to the baby. Strict asepsis during delivery will, to a large extent, prevent intra-natal infection.

The influence of care on the welfare of prematures is shown by the relative mortalities of legitimate and illegitimate premature infants, as illustrated by the following statistics, given by Bakker, of Hamburg :—

TABLE CLVII.

Weight	Mortality during the 1st year	
	Legitimate	Illegitimate
1,000-1,500 grm.	99	100
1,500-2,000 „	30	47
2,000-2,500 „	20	40

The results of proper ante-natal and neo-natal care on the incidence and mortality of prematurity is illustrated by the following sets of statistics :—

TABLE CLVIII.

(1) **New York Statistics.**

Cause of death	New York City, 1920 (no ante-natal supervision).	Maternity Centre Association, New York City, 1919-1921 (ante-natal supervision)
Stillbirths	46·5 per 1,000 births	25·1 per 1,000 births
Prematurity	14·7 „ „	4·8 „ „

TABLE CLIX.

(2) **Massachusetts.**

	Massachusetts as a whole	Cases without ante-natal care (1,800)	Cases with ante-natal care (2,100)
Stillbirths	34·7 per 1,000	35 per 1,000	22·7 per 1,000
Infant death-rate during first two weeks	32·4 „	32 „	14 „

Statistics of Prematurity (based on the figures for 1923).

(1) Incidence per 1,000 total live births = 50
(2) Mortality per 1,000 total live births = 23
(3) Neo-natal mortality per 1,000 total live births ... ... = 19

(4) Mortality per cent. of total infant mortality $= \frac{23}{69} \times 100$ ... ... = 33·3 per cent.

(5) Neo-natal mortality per cent. of total neo-natal mortality $= \frac{19}{32} \times 100$... = 60 per cent.

(6) Mortality per 1,000 premature births = 500

(7) Mortality of infants other than premature (per 1,000) ... ... = 46

(8) ∴ Mortality of prematures = ten times mortality of normal infants.

(9) Mortality according to **age and weight** (Ylppö) :—

TABLE CLX.

Birth weight	Number born	Died at the age of						
		1 day	5 days	1 month	6 months	1 year	2 years	5 years
All infants ..	668	62 = 9 per cent.	120 = 18 per cent.	206 = 31 per cent.	275 = 41 per cent.	301 = 45 per cent.	315 = 53 per cent.	320 = 53·5 per cent.
600-1,000 grm.	37	14 = 38 per cent.	27 = 73 per cent.	31 = 84 per cent.	33 = 89 per cent.	34 = 94 per cent.	—	—
1,000-1,500 ,,	183	28 = 15 per cent.	57 = 31 per cent.	88 = 48 per cent.	111 = 61 per cent.	114 = 65 per cent.	117 = 67 per cent.	118 = 67 per cent.
1,500-2,000 ,,	240	16 = 7 per cent.	26 = 11 per cent.	56 = 23 per cent.	83 = 35 per cent.	96 = 45 per cent.	102 = 48 per cent.	—
2,000-2,500 ,,	208	4 = 2 per cent.	10 = 5 per cent.	31 = 15 per cent.	48 = 23 per cent.	58 = 34 per cent.	62 = 36 per cent.	—

(10) Mortality according to **weight and temperature** of body (1st month) :—

TABLE CLXI.

Birth weight	Temperature	
	37-35° C.	28·9-27° C.
	Neo-natal mortality	Neo-natal mortality
600-1,000 grm.	66·6 per cent.	100 per cent.
1,000-1,500 ,,	37·5 ,,	85·7 ,,
1,500-2,000 ,,	21·1 ,,	60 ,,
2,000-2,500 ,,	5·9 ,,	20 ,,

The lowest body temperature of a premature baby that survived is 25·6° C. (76° F.). The baby weighed 1,700 grm., i.e., 3¾ lb. (Ylppö).

(11) Mortality according to **fœtal age** (Potel) :—

TABLE CLXII.

Age	Number	Number dying	Per cent. saved
6½ fœtal months	56	45	20
7 ,, ,, ...	131	76	42
7½ ,, ,, ...	53	17	70
8 ,, ,, ...	110	39	65
Total ..	350	177	50

(12) Mortality according to **length** :—

TABLE CLXIII.

Length in cm.	Viability per cent.
40	21
41	20
42	25
43	28
44	51
45	50
46	55
47	58

(13) Mortality according to **cause** (Francillon) :—

TABLE CLXIV.

Cause	Number	Mortality per cent.
Induction	28	21·4
Twins	49	14·2
Albuminuria	23	13
Syphilis	75	10·6
Heart disease	13	7·7
Unknown causes	499	2·7

Williams, in a series of 334 premature labours, found the following percentages :—

Syphilis	40	per cent.
Toxæmia	8	,,
Placenta prævia	[illegible]	,,
Fœtal deformities	3·3	,,

TABLE CLXV.—CAUSES OF PREMATURITY (Ylppö).

Cause	Number	Incidence	Survived 1 year
I. Maternal diseases—			
Syphilis	26	3·9 per cent.	27 per cent.
Tuberculosis	12	1·8 ,,	66 ,,
Other infectious diseases (influenza, pneumonia, &c.)	7	1 ,,	47 ,,
Eclampsia	21	3·1 ,,	52 ,,
Albuminuria	16	2·4 ,,	37·5 ,,
General constitutional disease (heart, diabetes)	13	1·9 ,,	46 ,,
II. Habitual familial prematurity	4	0·6 ,,	75 ,,
III. Anomalies or disease of maternal genital organs or passages (contracted pelvis, fibroids, placenta prævia, &c.)	30	4·5 ,,	43 ,,
IV. Trauma (fall, blow, &c.) ..	30	4·5 ,,	77 ,,
V. Twins (frequency in normal births = 1·3 per cent.)	128	19·2 ,,	63·5 ,,
VI. Triplets	12	1·7 ,,	33 ,,
VII. Unknown causes	369	55·2 ,,	49 ,,
Total	668	100 ,,	46 ,,

Incubators.

Historical.—Aristotle[1] refers to artificial means of hatching birds' eggs as a substitute for maternal incubation. "Eggs," says he, "are hatched with the same celerity spontaneously in the ground as by incubation. Wherefore in Egypt it is the custom to bury them in dung covered with earth." The Empress Livia is said to have carried an egg in her bosom until a chick was produced from it (William Harvey). The use of incubators for experimental pathological observations is also recorded in the Talmud.[2]

According to Baillet,[3] a certain Fortunio Liceri was born prematurely and was no larger than the palm of a hand, but his father, being a physician, "undertook to finish Nature's task and to work at the formation of the child with the same skill that men exhibited in hatching chicks in Egypt. He instructed a

[1] "Hist. Anim.," Lib. iv, cap. 2; Plin. "Hist. Nat.," Lib. x, cap. 54 (quoted by William Harvey, "On Generation," Sydenham Society Translation, London, 1847, p. 220).

[2] See W. M. Feldman, "The Jewish Child," London, 1917, p. 376.

[3] "Decision of Savants," Paris, 1872. For reference see Hess: "Premature and Congenital Diseases of Infants," 1924.

nurse in all that she had to do in the maintaining of exactly measured artificial heat and the requirements for his general care and feeding. He lived to be 79 years of age, and distinguished himself in science by a large number of works."

According to Hippocrates (460 B.C.), no fœtus born before the seventh month of pregnancy can be saved, although the Talmud states that an infant born at six and a half months can live. According to Mauriceau (1721), it is so rare to see a "7 months' baby" live, that scarcely one in a thousand escapes.

FIG. 136.—Double-walled tub.

The first incubator for infants was described by Denucé, of Bordeaux, in 1857, although, according to Clementovsky (quoted by Hess), Ruhl, of St. Petersburg, used a double-walled tub, the space between the walls being periodically filled with hot water (fig. 136), as early as 1835. Using a similar apparatus, which he called the incubating bed, Credé published results which show that by means of such a tub he was able to reduce the mortality of premature infants by 18 per cent., between 1860 and 1866.

In 1879, Winckel used a permanent bath (containing water at 96° to 100° F.), in which the infant floated. This was an

attempt to imitate intra-uterine conditions, but, owing to the danger of drowning and infection, it proved unpopular.

The first incubator, on the model of chicken incubators, was used by Tarnier, in the Maternity Hospital of Paris, 1881.

Tarnier compared the results obtained by his incubator with those obtained by Credé's bed incubator (*berceau incubateur*), and gives the following figures :—

TABLE CLXVI.

Weight of infant	Results : Difference between Credé and Tarnier
1,000-1,500 grm.	13·3 per cent.
1,501-2,000 ,,	9·8 ,,
2,001-2,500 ,,	1·5 ,,

FIG. 137.—U-shaped hot-water bottle for placing round the sides and feet of the baby to keep it warm.

Hutinel and Delestre state that in the Foundling House, in Paris, during the first five years after the installation of incubators the mortality amongst prematures went down from 66 per cent. to 36 per cent. It must, however, be remembered that contemporaneously with the incubators certain other improved hygienic precautions were instituted, viz., improved methods of feeding and of cleanliness. (For further details about incubators, see Hess's book on "Premature Infants.")

Personally, I believe that as good results—if not better—can be obtained without the use of incubators, especially of the closed variety, and I have reared the smallest premature baby recorded without such an apparatus. This is so because, on account of the high temperature and imperfect ventilation inside these apparatus, not only is the baby's metabolism retarded but germs develop within the apparatus with great rapidity, so that the infant has a greater chance of infection than when kept suitably wrapped up in a warmed cot. Another point against incubators is that they shut out the valuable ultra-violet rays of the sun which have such an important influence upon the growth and welfare of all infants. A useful apparatus for keeping the baby warm is illustrated in fig. 137.

If the baby progresses satisfactorily and has reached a weight of about 5 lb., the precaution with regard to temperature preservation need not be so rigorously observed. It may then be put in front of an open window on a sunny day, and after a day or two on a balcony or in the garden on a warm summer day. Gradually the baby may be accustomed to the usual routine of a normal baby, and in view of the great prevalence of rickets amongst them, such babies should have daily exposures to ultraviolet rays (mercury quartz vapour lamp), starting with two minutes at 90 cm., and going on to thirty minutes at 75 cm.

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CHAPTER XXII

HYGIENE OF THE TEETH

"Bid them wash their faces,
And keep their teeth clean."
SHAKESPEARE ("Coriolanus," ii, 3).

Dentition.—During the life-time of every individual who has attained adult age there erupt two separate sets of teeth, viz., the temporary teeth and the permanent teeth.

Temporary.—The temporary teeth are known also as milk or deciduous teeth. They begin to form at about the seventh month of fœtal life, and can be seen inside the jaws at birth by means of the X-rays (fig. 138). They do not, as a rule, make their appearance beyond the gums before about the sixth post-natal month, the periods of eruption of the various milk teeth, of which there are twenty in number, ten upper and ten lower ones, being as follows:—

2 lower central incisors	6- 8 months
4 upper incisors	8-12 ,,
2 lower lateral incisors	12-18 ,,
4 premolars	12-18 ,,
4 canines	18-20 ,,
4 second molars	24-30 ,,
20	

With the sole exception of the lateral incisors, all the lower teeth appear before the corresponding upper ones.

There is, however, no strict constancy, either with respect to the time or the order of the eruption of the teeth, although, if there is very great delay, rickets, or malnutrition, or cretinism may be suspected. It must be remembered, however, that one is not justified in ascribing delayed dentition to rickets unless there are other clinical signs of the disease, or there is X-ray evidence of it in the bones. Precocious dentition may also occur, and indeed in some cases babies are born with one or two of the lower incisors protruding from the gums. Louis XIV of France and Richard III of England were born with teeth, as was also Ludwig XIV, and the author has come across a couple of such

cases in his own practice. If they are loose in the jaw or interfere with proper nursing (biting of breast) they should be removed, but it must be remembered that once they are removed they will not be replaced until the permanent teeth make their appearance.

Congenital syphilis produces a tendency to early dentition, but teething at the age of four months may occur in perfectly normal and healthy babies. Teeth which erupt before the fourth month as a rule have as yet an insufficient coat of enamel, and are therefore liable to early decay.

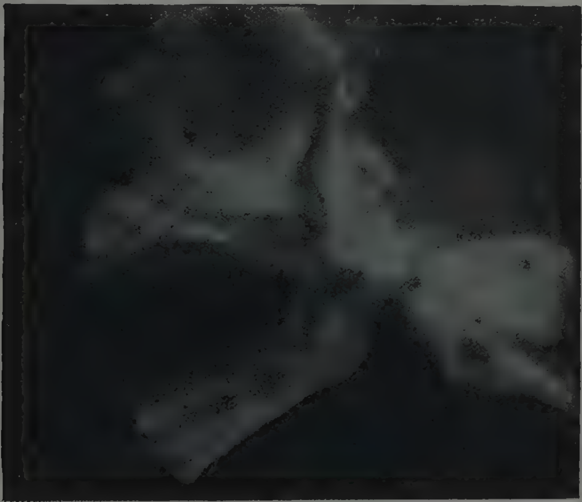

FIG. 138.—X-ray photograph of the skull of a baby at birth, showing the unerupted teeth, still incompletely calcified, inside the jaws. (From Symington and Rankin's "Atlas of Skiagrams," with permission of Longmans Green and Co.)

As a general rule breast babies cut their teeth earlier than those fed on the bottle.

Symptoms of Dentition.—Teething has long been regarded as a cause of many, if not of all the ailments of infancy. According to Hippocrates, dentition is ushered in by itching of the gums, fever, diarrhœa, and convulsions. Later authors wrote in the same vein, but were not in agreement as to which teeth were the most troublesome. Some blame the incisors because they are the sharpest, others the molars because they are the

bluntest. In the sixteenth century Ambroise Paré, the famous French surgeon, was searching for the cause of death in the 8-months' son of the Duc de Nivers, and found nothing until he opened the gums and discovered "all his teeth as if they were in array ready to come out," and he concluded that had lancing of the gums been done when the child lived, "doubtless he would have been saved." Paré, therefore, introduced this operation as a cure for all the troubles that were believed to be incidental to dentition (see p. 46). Of course, had Paré taken the trouble to examine the jaws of any dead new-born baby, he would have found all the teeth there and almost ready to come out, as can be verified nowadays by means of X-ray examination (see fig. 138).

On the other hand, of recent years there has been a tendency for the pendulum to swing too far in the opposite direction and to regard dentition as a purely physiological act which is unaccompanied by any constitutional disturbance in the baby. Neither of these extreme views is correct. Whilst in the majority of cases of well-nourished infants dentition causes very little inconvenience, it may in some cases be accompanied by some symptoms such as nasal catarrh (when the upper incisors appear), pyrexia, diarrhœa or vomiting, even in breast-fed babies. In rickety or syphilitic babies, as well as in those coming from a nervous stock, the appearance of each tooth may be heralded by a convulsion. In most babies there is some temporary standstill in weight, dribbling at the mouth, restlessness and disturbed sleep on account of pain in the gums, and the babies put their fingers in to alleviate the pain by rubbing the red and inflamed gums.

Treatment.—If the baby shows any of the above symptoms and the gums are seen to be red and swollen, they should be rubbed with a thoroughly cleaned finger three or four times a day. In addition the child should be given a bone ring to bite.

Permanent.—The permanent teeth which begin to be formed before birth are thirty-two in number, and they appear in the following order:—

First permanent molars	6 years
Lower central incisors	7 ,,
Upper incisors	8 ,,
Lower lateral incisors	8 ,,
First premolars (bicuspids)	9 ,,
Second premolars	10 ,,
Canines	12 ,,
Second permanent molars	12-15 ,,
Third permanent molars (wisdom)	17-25 ,,

Calcification of the permanent teeth proceeds during infancy, so that a defective diet may injuriously affect their development even before eruption.

[Many intelligent people in ancient times were ignorant of the number of teeth they had. The Talmud (Sanhedrin 39a) tells how R. Gamaliel once rebuked a man who bragged that he could count the number of the stars in the sky, but who, he found, could not tell the Rabbi how many teeth he had in his mouth. "If you do not know what is in your own mouth, said the Rabbi caustically, "how can you expect to know what is up in the sky?"]

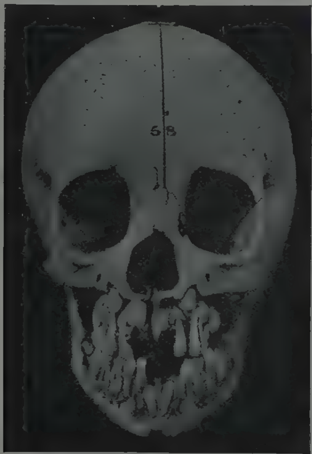

FIG. 139.—Skull of a child, aged 6 years, in which portions of the jaws were chiselled away to show the permanent teeth. Some of the temporary teeth are missing. (Museum of the Royal College of Surgeons.)

The Importance of the First Permanent Molars.—The first permanent molars or, as they are sometimes called from the date of their eruption, "the sixth-year molars," are in some respects the most important of all the permanent teeth. It is generally recognised that much of the physical deterioration occurring during the period of growth is directly or indirectly attributable to insufficient care and attention paid to the first permanent molar group. The first and second temporary molars are shed within a few months of each other, and in normal jaws the sixth-year molars soon appear in the space behind the last deciduous

molar. As therefore they erupt without pushing out temporary teeth, as is the case with the other permanent teeth (see fig. 139), they are often mistaken for temporary teeth, and are in consequence neglected until it is too late to save them. As, however, these teeth are the most important masticators, and as moreover they determine the shape of the jaw and the spacing of the other teeth, it will be realised that their loss is a considerable disaster from the point of view of the future welfare of the child. They may be distinguished from temporary teeth by looking less white and pearly, and by the enamel being less translucent, but the safest way to avoid their loss is by consulting a dentist when there is the least suspicion of decay in any of the teeth.

Effect of Congenital Syphilis upon the Teeth.—Congenital syphilis does not affect the temporary teeth, but it does leave its mark on the permanent ones, especially the central upper incisors. These show a peg-shaped notch, first described by Sir Jonathan Hutchinson. These peg-shaped teeth, which are therefore called Hutchinson's teeth, used to be seen fairly commonly some years ago, but are very rarely seen nowadays. This condition is present even before the teeth have erupted, as has been demonstrated by means of the X-rays.[1] On the other hand, such teeth are not absolutely diagnostic of congenital syphilis, and a case of Mongolian idiocy who showed no clinical or serological evidence of syphilis, but who possessed typical Hutchinson's teeth, has recently been recorded.[2]

Third Dentition.—There is an old belief that a third dentition may appear in very old age. According to Dr. Kanner,[3] X-ray examination has proved this to be quite possible, and at least two undoubted cases of third dentition have been established.

Structure of a Tooth.—A tooth consists of three portions, viz. :—

(1) *The crown*, which is the part exposed beyond the level of the gums.

(2) *The root*, which is the portion embedded in the gums and jaw-bone, and has one or more fangs.

(3) *The neck*, which is the constricted portion intermediate between the other two.

The whole of the tooth, crown, neck and root, is largely com-

1 Stokes, John H., and Gardner, Boyd S., *Journ. Amer. Med. Assoc.*, January 6, 1923, p. 28.

2 A. J. Mebane, *Amer. J. Dis. Ch.*, 28, 1924.

3 Leo Kanner, "Folklore of the Teeth," *Dental Cosmos*, 1926.

posed of a hard, ivory-like material called *dentine*. This resembles bone in appearance and chemical composition, although it differs from bone in microscopic structure. The dentine of the crown portion is covered and protected by *enamel*, which is the hardest structure found in the body, whilst that of the neck and roots is covered by *cementum*. Each tooth contains a central hollow, called the pulp cavity, which contains a vascular sensitive substance known as the dental pulp, consisting of connective tissue cells, nerves and blood-vessels which enter the pulp cavity through the root canals. It is the exposure of the nerves in the pulp caused by caries that gives rise to toothache.

Prevalence of Dental Decay.—Much money is being spent by educational authorities on the teeth of school children. Sir George Newman, in his Report for 1922, finds that where no dental treatment is available, the teeth of 70 or 80 per cent. of school children show definite decay, and that where action has been taken the figures fall to 40 or 50 per cent. Similar figures have been published from other parts of the country.

The extent of dental decay in adults has been shown by Major Helliwell,[1] who found that between 68 and 78 per cent. of recruits (according as they were Category A or Category C) required dental attention to fit them for army purposes, and that a normal arch with no evidence of dental disease was found in only 2 per cent. of the men in Category A. We see therefore that dental caries is prevalent from childhood onwards.

Effects of Dental Caries on the Child's Health.—Whilst the harmful effects of dental decay on the health of the child have probably been considerably overrated, there is no doubt that apart from the severe pain, occasional abscess formation, and the absence from school for which it is responsible, it may form the origin of serious disease, not only in childhood but in later life. The sepsis originating from a hollow tooth often gives rise to enlargement of the glands of the neck, which in predisposed children may become tuberculous, and the absorption of toxins from decayed teeth may bring on an attack of rheumatism.

The inability to masticate properly may cause indigestion which, according to some, may lead to malnutrition and anæmia. "Masticate your food well with your teeth," says a Talmudic proverb, "and you will find it in your legs." Further, a decayed temporary tooth also interferes with the proper eruption and alignment of the permanent teeth under them, resulting in loss of

[1] *Lancet*, December 25, 1920, p. 968.

their biting power (because the grinding surfaces of corresponding teeth do not meet properly) as well as in their decay.

Opinions are not quite unanimous regarding the relationship between dental caries and general malnutrition. Sir George Newman and others find a high correlationship between the two; on the other hand, Emerson,[1] from a study of 602 children, failed to establish any relationship between defective teeth and underweight, and Burpitt[2] even finds the relationship to be of a negative kind.

But whether malnutrition or anæmia of childhood is caused by defective teeth or not, there is no doubt that caries of teeth ought, if possible, to be prevented and, when detected, remedied, if we want to avoid the numerous dangers of decayed teeth in adult life.

Causes of Dental Caries.—The dental clinics at school, whilst useful in detecting the mischief when it has already come, and in preventing it from spreading, are an expensive and unpleasant luxury. The proper way of dealing with school children's teeth is to prevent rather than remedy their decay. In order to be in a position to do this, however, it is of course necessary to know exactly what are the causes which bring it about. Unfortunately these are not at all clearly understood. There are two working hypotheses, viz.:—

(1) *Constitutional.*—According to this, decay of teeth occurs in children either hereditarily predisposed to it, or who are in a bad state of health.

(2) *Environmental.*—This hypothesis assumes that acid fermentation occurring in the mouth as the result of taking too much carbohydrate, especially of a soft or sticky nature, will dissolve away the enamel and expose the dentine, or living tissue of the tooth, to the invasion of micro-organisms. The chief advocate of the carbohydrate theory is Dr. Sim Wallace.

The truth, as always, probably lies between these two hypotheses. Properly spaced teeth, situated inside a mouth in which fermentation is efficiently kept in check, will probably remain healthy, even in children who are delicate or hereditarily predisposed to caries. On the other hand, teeth which are crowded, and in the spaces between which morsels of food can collect, but from which they cannot be easily removed, will, in a mouth in

[1] "Nutrition and Growth in Children," 1922, p. 31.

[2] Quoted by Cronk.

which acid fermentation is abundant, become carious, even in healthy children coming from families with healthy teeth.

Broderick[1] summarises the conditions causing caries as follows:—

(1) With intact enamel caries is impossible.

(2) In a condition of health enamel continues to harden as the result of the progressive deposit of lime salts, the storage of which in the body is kept up by the endocrine apparatus.

(3) If the endocrine balance is disturbed in childhood (or ante-natally, i.e., in the mother during pregnancy), the calcium storage in the body becomes depleted and caries will result on account of:—

(i) Lessened alkalinity of the saliva, directly promoting caries.

(ii) Lessened alkalinity of the blood (or a relative acidosis) causing absorption of calcium from the enamel for the purpose of neutralising the acidosis.

(4) Without this susceptibility, due to deranged endocrine balance, exciting causes such as carbohydrate fermentation, &c., will not produce caries.

It is probable, as May Mellanby has shown (see p. 353, fig. 86), that the disturbed endocrine balance might be set up by deficiency of vitamins, whether before or after birth.

Influence of Food on Dental Caries.—The following table (Table CLXVII), published by Hamer in his Report to the Board of Education for 1920, giving the percentage of school children with no dental caries before, during and after the war, shows a gradual deterioration of the teeth in the early years of the war, when there was a scarcity of sugar, as well as of butter, eggs, milk and animal fats generally.

It tends to prove that it is not consumption of excessive amounts of carbohydrates, but rather a deficiency of vitamins that is in some way associated with dental caries (see p. 353).

TABLE CLXVII.—PERCENTAGE OF ENTRANTS WITH NO DENTAL DECAY.

Years:	1913	1914	1915	1916	1917	1918	1919	1920
Boys	62·2	51·6	47·6	28·6	40·5	48·6	49·3	53·2
Girls	63·0	52·0	47·2	27·1	39·2	45·0	48·1	52·7

[1] *Lancet*, December 10, 1921, p. 1216.

Prevention of Dental Caries.—If, as is probable, the causes enumerated above are those which are in reality responsible for dental decay, then we can formulate the following principles for preventing this condition :—

(1) Encourage the proper and normal formation of enamel :—

(*a*) Ante-natally, by feeding the mother on food rich in calcium as well as in vitamins (cereals, wholemeal bread, milk, butter, eggs, green vegetables, &c.), throughout her pregnancy, since the temporary teeth begin to be formed in the fœtus as early as the seventeenth week, when the dentine of the incisors and canines is laid down, and some of the permanent teeth begin to be formed after the twenty-sixth week. It must be pointed out, however, that in the largest majority of infants (about 98 per cent., according to some), the newly-erupted teeth are perfect.

(*b*) Post-natally, by giving suitable food to the baby after it is weaned (see p. 450). It has been estimated that 9 per cent. of breast-fed children have defective first teeth as compared with 22 per cent. among those that were bottle-fed.

(2) Avoid the irregular and overcrowded eruption of the permanent teeth by :—

(*a*) Giving the infant and young child food containing a sufficiency of vitamins, and sufficiently hard to give a progressively increasing amount of exercise for its temporary teeth as soon as they appear. This will help the proper development of the jaws as well as the flow of saliva, whose alkaline reaction will neutralise acid fermentation.

(*b*) Discouraging the use of comforters and finger-sucking, which tend to deform the jaws. In France, Professor Pinard, in 1926, succeeded in carrying a motion in the Chamber of Deputies prohibiting the sale of the *sucette* or dummy.

(*c*) Remedying any nasal obstruction which, according to some, is responsible for defective growth of the jaws, although Sir Arthur Keith and Mr. George Campion believe that the association between adenoids and defective maxillary growth is not one of cause and effect, but are both the result of impaired endocrine activity (e.g., pituitary and sexual organs), possibly again the result of vitamin deficiency.

(*d*) Prevention and cure of dental caries in the temporary teeth, since decay during this period is believed to interfere with the regular and orderly eruption of the permanent teeth, thus predisposing the latter to decay. It is here that dental school clinics accomplish their useful function.

(3) Teach the child oral hygiene, by training it to use a toothbrush night and morning, to remove any remnants of food as well as specific germs responsible for caries. By the age of 5 years, and sometimes even earlier, a child can be trained to brush its own teeth.

The results of dental clinics at schools, where teeth are inspected and treated, are seen from the following table by Sir George Newman, in his Report to the Board of Education for 1922:—

TABLE CLXVIII.—PERCENTAGE OF SOUND TEETH IN CHILDREN, 12 YEARS OLD, IN LONDON.

Years:	1913	1915	1917	1919	1921	1922
Boys	50·0	53·1	55·2	53·1	60·3	65·3
Girls	52·4	55 5	56·4	56·5	63·5	67·5

It will be seen that the best results occurred between 1919 and 1922. This is due to the fact that during these years the children who first came under inspection and treatment in 1913 were leaving school.

(4) Promotion of good general nutrition (avoidance of rickets, anæmia, &c.) by means of general hygiene (fresh air and sunlight, &c.).

(5) Giving the child foods which: (i) do not contain excess of carbohydrates, especially sweets between meals; (ii) require mastication; and (iii) are tooth-cleansing. Such foods are fish, meat, vegetables, stale bread, fresh fruit, such as apples, &c.

(6) Every child, from the age of 3 or 4 years upwards, should have its teeth examined by a dentist at least once a year, and preferably twice a year, and any caries that may be detected remedied. As far as possible extraction should be avoided.

There is no reason why with proper care each person should not have a mouth full of teeth which, as described by King

Solomon, are "like a flock of sheep that are even shorn, whereof every one bear twins and none is barren among them."

Dental Hygienists or Dental Dressers.—With the object of minimising the expense in connection with the dental examination and treatment of school children, Dr. Alfred C. Fones, of Bridgeport, Connecticut, introduced the idea of training intelligent young women in the principles of oral hygiene, as well as in the methods of detecting and recording any dental decay or irregularity. These "dental hygienists" who have been drafted into a number of schools in many of the American States as well as in New Zealand, do the surface work and all the mouth sanitation at a fraction of the fees of registered dentists. Their

FIG. 140.—Dental Dressers at Work.

training lasts one year in the case of America and Derbyshire, and two years in New Zealand. They were also for a time introduced into certain parts of England (i.e., Derbyshire, etc.), but were discontinued on December 31, 1925, because the system was declared to be a contravention of the Dentists Acts.

As soon as the child enters school for the first time, its mouth is thoroughly examined by the dental hygienist, and any dental defect is referred to the school dentist for treatment. At the same time she cleans the child's teeth and gives group and individual instruction in oral hygiene, emphasises the importance of clean and healthy teeth, and conducts toothbrush drill. It has been estimated that 1,000 dental hygienists and 100 regis-

tered dentists would be able to do the whole of the prophylactic and remedial dental work in all the London schools.

It is claimed that in those places where dental hygienists have been employed there has been a practical elimination of defects in the permanent teeth, and that their universal employment would rapidly and at small cost render dental school clinics unnecessary. Such a claim may perhaps be somewhat extravagant, but there can be very little doubt that their introduction into schools would not only save the ratepayers considerable sums of money, but would also greatly accelerate the reduction of dental defects in the school children.

Toothbrush Drill.—As soon as the child is able to understand and do things for itself, it should be taught to wash its teeth night and morning, especially at night, so as to prevent the decay of food material remaining in the interdental spaces overnight. The brushing should be done up and down, and from side to side, both on the front and back surfaces of all the teeth. The brush, which should have bristles adapted to enter the spaces between the teeth —so as to have a "toothpick" action—as well as a projecting tuft at the end which will go round behind the back molars, should then be rinsed and put out in the rack-stand, preferably in the sun. Any simple tooth powder, such as finely-powdered precipitated chalk, will do, and after the brushing the mouth should be rinsed with water. This toothbrushing should be insisted on until it is established as a regular habit.

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CHAPTER XXIII

HYGIENE OF THE SENSE ORGANS

> "There is no sense that has not a mighty dominion, and that does not by its power introduce an infinite amount of knowledge. If we were defective in the intelligence of sounds . . . it would cause an imaginable confusion in all the rest of our science. . . . Let an understanding man imagine human nature originally produced without the sense of seeing and consider what ignorance and trouble such a defect would bring upon him. What a darkness and blindness of soul. . . . We have formed truth by the consultation and concurrence of our senses."—MONTAIGNE.

THE anatomy and physiology of the child's eyes and ears have been fully dealt with in my "Child Physiology," and the hygiene of these organs is described in considerable detail in most books on school hygiene. It will therefore suffice if I give here an epitome of the subject, pointing out how far the special anatomy of the child's sense organs helps one to understand the principles of hygiene upon which the prevention of diseases of these organs—especially those leading to deafness and blindness—are based.

The Ears.

The parts of the ear which interest us here are :—

A. The Sound-conducting Part:
- (*a*) The external auditory meatus.
- (*b*) The tympanic membrane or drum membrane.
- (*c*) The middle ear, or tympanum or drum.

B. The Sound-perceiving Part.—The internal ear.

Peculiarities of the Child's Ear.

The external auditory meatus is almost entirely occluded at birth, and hence new-born babies cannot hear until the meatus becomes canalised shortly after birth.

The tympanic membrane is nearly horizontal at birth, its inclination being approximately 12½ degrees; the inclination in the adult being approximately 45 degrees. It is also comparatively thicker in the infant than in the adult. Both these peculiarities of the membrane and external auditory meatus in early life are of

importance. Otoscopy, which, as we shall see later, is important for the detection of unsuspected ear disease, is very difficult in infants on account of the narrowness of the external auditory meatus as well as the nearly horizontal position of the membrane. The greater thickness of the membrane makes it more difficult for any pus in the middle ear to break through the membrane and escape outside, and hence allows the pus to spread to more vital parts, such as the brain. Hence, we find that whilst the total number of deaths from ear and mastoid disease during 1923 was 1,162, the number of deaths from these causes during the age-period 0-5 years was no less than 277, i.e., nearly 25 per cent. of the total, and that during the first year of life 127, or nearly 11 per cent.

The Tympanic Cavity or Middle Ear.—The upper wall of this cavity, which is formed by the union of two bones, the petrous and squamous bones, is characterised in the infant by the fact that at birth the line of union is not complete, so that in early life, up to about 6 years of age, there is greater freedom for pus or inflammatory material to pass from the ear to the brain, from which the upper wall separates the middle ear. In spite of this, however, middle-ear suppuration does not travel up to the brain as often as one would expect it to do, because the presence of the petro-mastoid fissure in the infant gives the pus an exit to the subcutaneous region of the mastoid. The tympanic cavity contains the ossicles whose vibrations in unison with those of the tympanic membranes are conveyed to the internal ears where the sound impressions are made and whence the impressions are carried by the auditory nerve to the brain.

The Eustachian tube which connects the middle ear with the naso-pharynx, and effects an exchange of air between the atmosphere and the ear, is practically cylindrical throughout its course in the infant, instead of being of variable diameter, as in the adult. It is also shorter, wider, and opens into the naso-pharynx nearer the hard palate than in the adult, the comparative measurements being as follows:—

TABLE CLXIX.

Age	Length	Width	Distance from hard palate
At birth	20 mm.	3 mm.	6 mm.
2 years	33 ,,	2 ,,	9 ,,
6 years	36 ,,	1 ,,	11 ,,
Adult	38 ,,	1 ,,	13 ,,

Hence the younger the child the easier it is for naso-pharyngeal infection to reach the middle ear through the Eustachian tube. For all these reasons middle-ear disease and its consequences are commonest in early life.

The mastoid process is absent in early infancy and only begins to develop when the sterno-mastoid muscles grow in size, i.e., when the infant begins to balance its head properly. The antrum is relatively large and is more superficial, and contains no definite mastoid cells.

Results of Obstruction of the Eustachian Tube.—If, as the result of adenoids or other chronic naso-pharyngeal catarrh, the Eustachian tube becomes obstructed, the oxygen of the air in the middle ear gradually becomes absorbed and, in consequence of the rarefaction of the air within the cavity, the tympanic membrane becomes drawn in, its vibrations are hampered and hearing becomes impaired.

The peculiarities of the internal ear are more of physiological than hygienic interest, and are discussed fully in the author's "Child Physiology."

TABLE CLXX.

Age	Number of deaths
0-1 year	127
1-5 years	150
0-5 ,, ...	277
5-10 ,, ...	141
10-15 ,, ...	139
15-20 ,, ...	113
20-25 ,, ...	85
25-30 ,, ...	71
30 onwards	336
Total ..	1,162

Frequency of Ear Diseases in Childhood.

(*a*) *Mortality.*—During 1923 the total number of deaths in England and Wales from diseases of the ear and mastoid sinus was 1,162. In proportion to the total population, therefore (40,000,000), as well as to the total number of deaths (about 450,000), the figure is comparatively insignificant, representing as it does a mortality rate of 0·029 per 1,000, and a proportional mortality of 0·25 per cent. of the total mortality. Nevertheless,

the figure is very high in comparison with the total number of deaths (viz., 70) that resulted in the same year from diseases of the neighbouring sense organ, the eye.

The age distribution of deaths from ear diseases, is given in the **preceding table** (Table CLXX).

(*b*) *Morbidity.*—It is impossible to get exact figures of the incidence of ear diseases in children, but of the children in elementary schools medically examined in 1923 about 8 per 1,000 suffered from middle-ear disease, corresponding to a total of 46,000 school children with ear trouble in England and Wales during that year. In London the proportion was more than twice as high (17 per 1,000). But in view of the necessarily hurried nature of the examination, which can only detect obvious discharges from the ear and very marked deafness, as well as of the difficulty of otoscopy in young children, it is practically certain that the true incidence is considerably higher. This is confirmed by the fact that post-mortem examinations in children dying from diseases unconnected with the ear show evidence of purulent disease of the middle ear in a very large number of cases. Thus, in a series of 100 such examinations in children under 4 years of age, there was evidence of ear disease in 91, although in only 10 of these was the trouble recognised during life (Ponfink[1]). Indeed there is very little doubt that many of the cases of pyrèxia in children, without obvious cause, are due to middle-ear disease in which no other symptoms are present, as can be verified by a careful examination of the ear.

Defective Hearing.—But quite apart from the pain, suffering and death which may be caused by diseases of the ear, all pathological conditions of that organ are important on account of the impairment of hearing which may result from them. Numerous investigations by different observers show that about 25 per cent. of all children have defective hearing of various degrees, and that in 5 per cent. of the children the hearing is sufficiently bad as to interfere with their school work.

The normal range of perception of sound is between 20 and 20,000 vibrations per second, but in the course of ordinary conversation a range of no more than about 400 vibrations, viz., between 120 and 520 vibrations per second, is employed. Hence it is possible for a child to have considerable impairment in hearing before such defects are suspected. From Table CLXXI

[1] See J. A. Colliner, *Arch. of Pediatrics*, 1916, p. 434.

we see that the majority of cases of incurable deafness could have been prevented had they been detected early enough.

Causes of Defective Hearing.—The following table, compiled by Macleod Yearsley, gives the frequency of the various causes of deafness in 1,863 L.C.C. school children :—

TABLE CLXXI.

Causes	Frequency
1. Congenital	654
2. Acquired :—	
(*a*) Infections, e.g., measles, diphtheria, &c.	408
(*b*) Primary middle-ear disease ..	448
(*c*) Nervous (sequela of meningitis, &c.)	188
(*d*) Other causes (injury, &c.) ..	165

The following table represents the ages at which deafness manifests itself in New York :—

TABLE CLXXII.

Age	Percentage
Born deaf	30·6
0-3 years	30·4
3-6 ,, ...	23·0
After 6 years	16·0

From these tables we learn that not only can the majority of cases of deafness be prevented, but that the care of the ears must begin before the child reaches school age. Indeed, preventive measures must begin even before the child is born or conceived, since, of the congenital causes, some are probably due to parental syphilis, and in others, viz., about 15 per cent. of all congenital cases, the deafness behaves like a transmissible Mendelian recessive character, so that the birth of such children could, to a large extent, be prevented by the application of the appropriate eugenic rule (Rule 2, p. 228). It has been estimated that some 10,000 people who are themselves not deaf are normal heterozygotes, who are thus able to transmit deafness if they mate with other heterozygotes or recessives.

Anatomically, the causes of deafness are :—

(*a*) Those which interfere with the sound-conducting parts, e.g., (i) obstruction of the external ear, such as by wax, foreign body, &c. ; (ii) disease of the middle ear ; (iii) obstruction of the Eustachian tube.

(*b*) Disease of the internal ear.

(*c*) Disease of the auditory nerve.

(*d*) Disease of the brain.

Most of the causes coming under (*a*) are preventable and curable. Most of those coming under (*b*), (*c*), and (*d*) are neither preventable nor curable.

Auditory Acuity.—The hearing of each ear should be tested separately, by whispering the numbers from 20 to 100. The normal ear hears whispering of the vowel sounds at the following distances :—

a	e	i	o	u	
26	38	34	25	11	metres,

and of ordinary words at 25 metres. In schools, however, it is not practicable to use a longer distance than 6 metres, but it is almost certain that at this distance many early cases are missed. In order to use the same intensity of whisper, the forced expiratory whisper is used, and the child should have its eyes shut during the test so as not to recognize the sound from the movements of the lips.

Degrees of Deafness.—All varieties of deafness are encountered in early life. Of those children that are so deaf as to require education in special schools, about 50 per cent. owe their condition to preventable middle-ear disease, and 33 per cent. are of congenital origin. Of the minor cases of deafness all are due to middle-ear disease.

Prevention of Deafness and of other Ear Diseases.

(*a*) *Ante-natal Measures.*—A considerable proportion of congenital deafness may be prevented by :—

(i) Eugenic means before marriage, such as the avoidance of marriages between two recessives—marriages which are quite unjustifiably contracted in the case of inmates in the asylums for the deaf—as well as between a recessive and a probable heterozygote, or between two possible heterozygotes. Consanguineous unions in the cases where congenital deafness exists in the family should therefore be discouraged. Also, people with uncured syphilis should not marry, since

a certain number of congenital deaf mutes owe their condition to parental syphilis.

(ii) Proper ante-natal treatment of syphilis.

(*b*) *Post-natal Measures.*—To prevent deaths from ear diseases, as well as the various degrees of deafness resulting from such diseases in early life, it is necessary to get hold of the baby right from the time of birth. Every infant welfare medical officer must have special training in aural diseases, and facility for the examination of the ears and naso-pharynx of all babies under his or her supervision. Such examination should be continued during the pre-school period. In addition, it is necessary to prevent nasal catarrh, which is best done by giving the children an open-air life. In open-air schools nasal catarrh is practically absent, whilst in the ordinary schools a large proportion of the children are affected by it. The prevention of, and proper attention during attacks of, infectious diseases will eliminate all the cases of death or deafness due to this cause. The primary ear diseases are caused by septic tonsils and adenoids, as well as by dental caries. Indeed, according to Edwards, "there is a close relationship between the condition of enlarged tonsils in childhood and the carious state of the teeth," and he believes that enlargement of tonsils is due to dental decay. It is, however, practically certain that although the two conditions are frequently associated, the one is not due to the other, since many cases of dental caries occur without tonsillar enlargement, and vice versa. Thus, large tonsils and adenoids are frequent in infancy before dental caries can occur. *Adenoids should be removed if they interfere with proper nasal breathing or cause trouble in the ears.* This operation may have to be done in babies who cannot take the breast or bottle properly and have to stop frequently in the middle of a feed to take a breath. Children with the typical adenoid face, viz., those that keep their mouths constantly open and snore at night, may also require to have their adenoids removed by operation.

The Question of Operation for Tonsils and Adenoids.—Whilst there can be no doubt that all children are better off without their adenoids as regards their ears, and the possibility of rheumatic infection without their tonsils, opinions are not in agreement with regard to their indiscriminate removal in every case of enlargement. Zahorsky, for instance, believes that the tonsil plays a rôle in the production of immunity, and Blum has produced statistics to show that tonsillectomy favours the occurrence of respiratory

diseases. But whether this be so or not, there is equally no doubt in the author's mind that the operation should not be undertaken too lightly, and one should carefully weigh the arguments for and against operation in every case. Fatal accidents (due to status lymphaticus) during the operation are admittedly rare, but they do occur, even when the operation is performed by the most skilful surgeon, and nobody can foretell which are the cases that are going to turn out unfavourably. Every case of tonsils and adenoids should be brought to the attention of the doctor, who should decide regarding the advisability of operation. (See preceding paragraph.)

Prevention of Adenoids.—It is believed by some that enlargement of adenoids is produced by chronic nasal catarrh, and that the prevention of the latter will also prevent the former. To prevent catarrh it is recommended that not only should contact with sufferers from nasal catarrh be avoided, but that the feet should be properly shod to protect against damp, and that the child should live an outdoor life as much as possible. In addition, "nose-blowing drill" and **deep-breathing exercises**, with the mouth shut during inspiration and expiration, should be practised. Dr. Octavia Lewin calls chronic nasal catarrh "constipation of the head," since it results in the prolonged retention of waste in the head, and its prevention consists in thorough blowing of the nose every morning to clear it of the mucus that has accumulated during the night. Sniffing should not be permitted, since by so doing the secretions are aspirated into the naso-pharynx and the Eustachian tubes. Sneezing into handkerchiefs should be encouraged, and may even be artificially produced by the application of a snuff made from powdered orris root and soap. Large and even slightly septic tonsils can sometimes be cured by daily applications of a preparation of iodine, e.g., Mandl's paint. It is when such preventive measures fail, or when the tonsils and adenoids actually cause symptoms, that the question of operation is to be considered.

Education of the Deaf Child.

(1) If the child is only slightly deaf, it is sufficient to place him in front of the class, which will not only enable him to hear better but will also enable the teacher to watch the child more closely to be sure that he hears all that is said.

(2) Macleod Yearsley recommends that all the moderately deaf children should be segregated in a special school under the care of a

special teacher, who will devote one-third of the school time to instruction in lip-reading. "Thus they obtain practice in lip-reading and learn to combine it with what remains to them of hearing." As the deafness subsequently increases or decreases they will make more or less use of lip-reading.

(3) Congenital deafness, or deafness acquired before the child has learned to speak, must begin to be dealt with as early as possible. Under the present law a hearing child is compelled to begin school at 5, and a deaf child need not attend until the age of 7. This is much too late an age. Training should begin at an age when the brain and speech organs are in the most plastic condition, and hence the sooner the child can be sent to school the better. Congenital deafness should, according to Somerville Hastings, be made a notifiable disease, and school attendance of children so afflicted should begin at 3 years of age.

The Eyes.

It has been estimated that some 90 per cent. of our experiences are obtained by means of the eyes, and yet Helmholtz, the greatest expert on the physics and physiology of vision that has ever lived, is credited with the remark that he would have been ashamed to have turned out so imperfect an optical instrument as the eye. From the standpoint of the scientific instrument maker, the criticism is probably a just one. The eye is primarily intended to serve as a photographic camera, in which images of external objects are very accurately focused by a system of lenses upon a sensitive plate, and hence for the eye to fulfil this function perfectly, the sensitive plate (i.e. the retina) ought to be at such a distance from the focusing part that clear images of external objects are focused upon it without any conscious effort. This, as we shall see, is not the case. Further, the eye, contrary to the best instruments, has no arrangement for eliminating chromatic aberration, that is, the different focusing of the different colours of white light, each of which is refracted to a different extent, the blue rays being brought to a focus nearer than the red. Moreover, the normal eye does not rival in acuity of vision an ordinary magnifying glass.

Nevertheless, the eye is in most cases a nearly perfect instrument. Indeed, its comparatively low visual acuity is an advantage rather than a failing, and the chromatic aberration is, under ordinary conditions, not noticeable. If our eyes were as

keen as microscopes, everything, including the most finely polished crystal and the smoothest skins in our own bodies, would look rough and uneven. Indeed, Sir R. Steele describes the wonderful mechanism of the eye as follows: "As our greatest pleasures and knowledge are derived from the sight, so has Providence been more curious in the formation of its seat, the eye, than in the organs of the other senses. That stupendous machine is composed in a wonderful manner of muscles, membranes, and humours. Its motions are admirably adapted by the muscles; the perspicuity of the humours transmit the rays of light; its rays are regularly refracted by their figure; the black lining of the sclerotes effectually prevents their being confounded by reflections. It is wonderful indeed to consider how many objects the eye is fitted to take in at once and successively in an instant, and at the same time make a judgment of their position, figure and colour."

The Gross Structure of the Eye.—A perfectly-shaped eye probably does not exist. It is approximately, but not quite, spherical in shape, and the system of refractive media within it is so arranged that when the eye is at rest distant objects are sharply and perfectly focused upon the retina without any conscious effort on the part of the eye muscles.

The eyeball has three coats, external, middle and internal, containing within them various structures for focusing light upon the sensitive internal coat (the retina), for regulating the intensity of the light falling upon that coat, for transmitting the impressions received by the sensitive retina to the brain where that image is perceived and interpreted. It also has a system of muscles, voluntary and involuntary, for the movement of the eyes in different directions, as well as for altering the radius of curvature of the refracting surface to accommodate the eye to vision of objects situated at different distances from it. (See fig. 141.)

A. The Coats.

(1) The *outer coat* consists of two parts, viz.: (*a*) The white of the eye, known as the *sclerotic*, which is tough and opaque and forms the greatest part (between one-sixth and one-fifth) of the eyeball. (*b*) The protuberant transparent part, forming between one-sixth and one-fifth of the surface of the eyeball, and known as the *cornea*. The cornea and visible part of the sclerotic is covered by a delicate membrane, called the *conjunctiva*, which is reflected on to the internal surface of the upper and lower eyelids.

(2) The *middle coat* also consists of two parts, viz.: (*a*) The *choroid*, which lines the sclerotic. It is thick and vascular, and contains dark pigment to prevent the light which enters the eye from being reflected in various directions, thus dazzling the eye. It thus corresponds to the black lining of the photographic camera. In albinos, in whom pigment is defective, there is the "pink eye," because of the pink reflection from the intra-ocular blood-vessels. (*b*) The *iris*, which is the continuation of the choroid in front of the corneo-sclerotic junction. This is a membranous curtain which lies in front of, but does not completely cover, the lens, and gives the characteristic colour to individual eyes. The circular hole in front of the lens is known as the pupil. By the contraction or relaxation of the involuntary muscle fibres in the iris, the pupil becomes smaller or larger. The iris therefore acts like the diaphragm in a photographic camera or microscope for regulating the amount of incoming light. The iris also, by cutting off the peripheral rays striking the lens, eliminates the effects of spherical aberration.

(3) The *internal coat*, or *retina*, is the delicate sensitive membrane which is the spread-out portion of the optic nerve which pierces the sclerotic and choroid at the back. It is upon the retina that visual images are thrown, and the impressions formed by these images are conveyed to the brain by the optic nerve.

B. The Contents.

(1) The *anterior chamber* is the cavity bounded by the cornea in front and the iris and pupil behind. It contains a watery substance, the *aqueous humour*.

(2) The *lens*.

(3) The *vitreous humour*, which is a jelly-like transparent structure filling the eyeball behind the lens. The chamber behind the iris containing the lens and vitreous is called the *posterior chamber*. It is the aqueous, the lens and the vitreous which form the wonderful refractive system mentioned above which focuses images upon the retina.

C. The Muscles.

(1) The *voluntary group of six muscles*, attached to the orbit or eye-socket at one end, and to the eyeball at the other. By their contraction they rotate the eyeball in every direction in space.

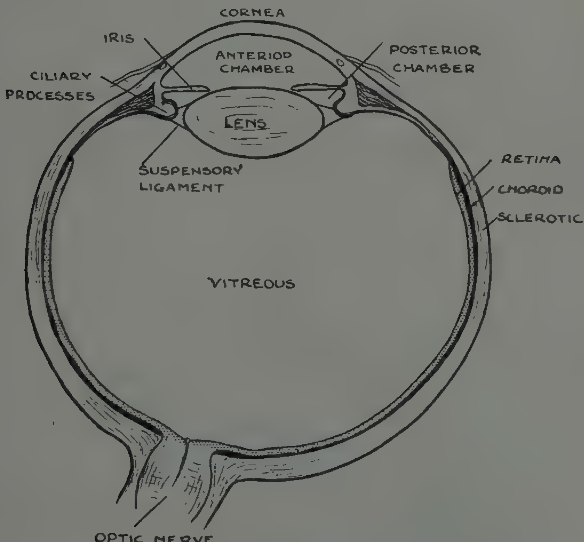
(2) The *involuntary ciliary muscles*, which by their contraction alter the curvature of the lens and thus accommodate the eye to see objects at different distances.

Peculiarities of the Child's Eye.—We have seen that the eye is not quite spherical. This imperfect sphericity is still more marked in the infant than in the adult, as can be seen from the following table:—

TABLE CLXXIII.

Diameter	Adult	New-born
Antero-posterior	22·85 mm.	16·4 mm.
Horizontal or transverse ..	24·43 ,,	16·0 ,,
Vertical	23·7 ,,	15·4 ,,

Hence the antero-posterior diameter is relatively to the other diameter greater in the new-born than in the adult.



[By kind permission of Messrs. Methuen and Co., Ltd.

FIG. 141.

The cornea is relatively larger at birth, when it forms about one-fifth of the whole surface of the globe, than in the adult, when it is no more than one-sixth of that surface. Indeed, the absolute size of the cornea is almost as large at birth as in the adult, a fact which explains the apparently great size of the child's eye.

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Opinions are not unanimous regarding the curvature of the cornea. Some believe it to be the same, and others think it to be of greater curvature than the adult's cornea.

The iris is less pigmented in the infant, hence the eyes of all young babies, even of negroes, look blue.

The pupil, in a state of rest, is smaller in the infant, it being about 1·8 mm. at birth and about 3·5 mm. in the adult.

The choroid has the same thickness in the new-born as in the adult.

The retina is thinner in the infant, but contains the same number of rods and cones (the sensitive cells) per unit area as does the adult's retina.

The Lens.—Whilst in the adult the radii of curvature of the anterior and posterior surfaces of the lens are 10 and 6 mm. respectively, they are approximately equal at birth, viz., 3·5 mm. in each case. The lens is also thicker in the infant, its thickness being 5 mm. as compared with 3·6 mm. in the adult, and it is also more elastic. The greater thickness of the lens at birth makes it protrude more prominently into, and diminish the depth of, the anterior chamber. The greater elasticity of the lens in early life renders accommodation, which depends upon alteration of lens curvature, easy.

The Conjunctiva.—In the adult the superficial layers of the conjunctiva are squamous or flat, and very horny and resistant to microbic invasion. In the infant the superficial cells are cubical and less resistant. Hence they are more penetrable by gonococci, a fact which explains the relatively greater frequency of gonorrhœal ophthalmia at birth than in the adult.

The Muscles.—Owing to the lack of ocular muscle balance in infancy, squinting is fairly common in babies, even with fairly normal vision.

Weight of the Eye.—The weight of the eye increases during growth from about 2·5 grm. at birth to about 7·5 grm. in the adult. During the same time the brain increases in approximately the same ratio, i.e., from about 400 to 1,500 grm.

The volume of the eye increases in about the same ratio as its weight.

Hygiene of the Child's Eyes.—The care of the eyes in early life is concerned with the prevention of :—

(*a*) External diseases.
(*b*) Defective vision.
(*c*) Squint.
(*d*) Injuries.

(*a*) **Prevention of External Diseases.**—The most important external disease is ophthalmia neonatorum. This has been fully dealt with in Chapter XI, pp. 315-17. Simple conjunctivitis blepharitis (inflammation of the eyelids) and styes in children, if acute, are due in many cases to uncleanliness and can therefore be prevented by ordinary clean habits. If the condition is chronic, it is frequently associated with defective vision and can be cured by correcting the child's vision. Interstitial keratitis is always of congenital syphilitic origin, and is therefore preventable. Phlyctenular conjunctivitis is almost certainly due to tuberculosis, since not only does it occur in poor, anæmic and under-nourished children, but in nearly 30 per cent. of the cases are there signs of tuberculosis in other parts of the body; in nearly 100 per cent. of cases is the von Pirquet reaction positive; the histological appearance is that of a tuberculide; and lastly, instillation of tuberculin into rabbits' eyes produces this condition experimentally, although other methods of producing it experimentally have failed. This disease is much less common amongst Jewish children, because they are well fed.

Xerophthalmia is another but much rarer condition which is preventable. It is a condition characterised by the appearance of white triangular masses of degenerated epithelium, placed with their bases against the edge of the cornea. The condition is associated with night-blindness (i.e., inability to see in reduced light), and may lead to softening of the cornea. It has been experimentally produced in rats by feeding on a diet deprived of vitamin A, and hence may be called a deficiency disease like rickets. It is prevented and rapidly cured by fresh milk and cod-liver oil.

Trachoma is another preventable disease, which is, however, very rare in this country. It is communicated from person to person by means of towels and can therefore be easily prevented.

(*b*) **Defective Vision.**—We have seen that the antero-posterior diameter of the normal adult eye (i.e., the line from the centre of the cornea passing through the centre of the lens to the retina) is about 23 mm. The various refractive media are so arranged that when the eye is at rest parallel rays (in practice, rays coming from an object more than 6 metres away from the eye) are focused exactly, and give a clear image upon the retina (fig. 142). When objects are nearer than 6 metres the rays, instead of being parallel are divergent, and the refractive media of the eye at rest are not sufficiently strong to bring them to a focus on the retina. To

meet this divergence the process of accommodation is made use of, whereby the ciliary muscles enable the lens to bulge forward, thus increasing the curvature of its anterior surface, and by so doing increase its refractive power to an extent sufficient to bring the divergent rays (which would otherwise meet beyond the retina) to a focus upon the retina.

The condition of normal vision is called *emmetropia*, and in such a condition, therefore, not only must the optic axis (i.e., the axial length) of the eye be 23 mm., but the refractive media must be clear and transparent and of normal curvature, and the retina, optic nerve and brain must be healthy.

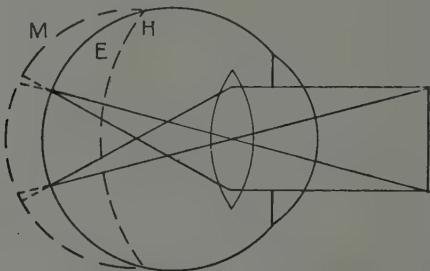

FIG. 142.—Path of rays in emmetropia, E. Myopic eye, M; and hypermetropic eye, H.

Ametropia.—If there is a disproportion between the focal length of the refractive system and the length of the optical axis (ametropia), the image formed on the retina will be blurred and indistinct. Ametropia may be of three kinds, viz.:—

(1) *Hypermetropia* or *long sight*, due to shortness of the optical axis (hypermetropia in Greek means "over-moulded eye"). In such a case parallel rays will, unless the process of accommodation is brought into use, be brought to a focus *behind* the retina. To enable such an eye to see properly without the exercise of accommodation, a convex lens of a strength varying with the degree of hypermetropia must be placed in front of it.

Hypermetropic children cannot do near work without considerable difficulty. Their eyes get red and ache, and they also get headache as the result of excessive strain used in the process of accommodation, and because they cannot apply themselves to their lessons without feeling the strain may be unjustifiably

punished for laziness. Moreover, the constant exercise of their accommodation, even for distance, is a great strain upon their nervous system. It has been estimated that 30 per cent. of school children in the United States are two years behind their natural grade, and about 10 per cent. of these owe their backwardness to defective vision. When the condition is remedied their progress improves.

(2) *Myopia.*—In this condition the axial length of the eye is too long, i.e., longer than 23 mm., so that images of external objects are formed *in front* of the retina. In order to see distinctly such children partially close their eyes (myopia in Greek means "closed eye") and thus practically convert their eyes into pinhole cameras, in which refraction is not required at all. Such eyes require concave lenses to render the rays divergent before meeting the eye, and thus bring them to a focus on the more distantly placed retina. The strength of the lens necessarily varies with the degree of the myopia. A myopic eye will also see quite well if a black disc with a small slit be placed in front of it. Owing to the fact that blue rays are brought by the eye to a focus nearer than the red, people are somewhat myopic in blue light and somewhat hypermetropic in red. The normal eye is best focused for yellow light (see fig. 143.)

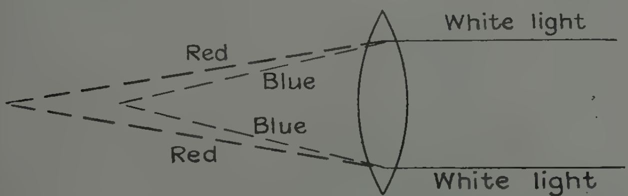

Fig. 143.—Effect of chromatic aberration.

Myopic children cannot see things well at a distance. They cannot read what the teacher writes on the board, nor can they recognise their friends across the street. They can, however, see quite well to read even the smallest print; indeed, they are able to do finer work than normal-sighted persons, because they bring the objects very near their eyes and therefore see them very large.

(3) *Astigmatism.*—In astigmatism the focusing power of the eye is not the same in all meridians, and the condition is due to the fact that the cornea, instead of being a segment of a perfect

sphere—so that its curvature, and therefore its refractive power, is the same in all meridians—is more curved in one meridian (usually the vertical) than in the meridian at right angles to it (the horizontal). Such an eye brings parallel rays to a focus nearer in the meridian of the greater curvature than in the other, and is therefore never able, even with accommodation, to produce a perfectly focused image on the retina (astigmatism in Greek means "no point" or "no focus"). If uncorrected by suitable lenses, astigmatism gives rise to eye-strain and headache, as well as to interference with school work. It requires, for its correction, a lens which is more curved in one direction than the other, i.e., either a cylindrical lens in cases where the curvature of the cornea is abnormal in one meridian only, or a combination of spherical and cylindrical lenses when the curvature is not normal in any meridian, but is greater in one meridian than in another. The greatest curved meridian of the lens is placed parallel to the least curved meridian of the cornea.

The Ametropia of Infancy and Childhood.—We have seen, on p. 543, that at birth the eye is short from before backwards. Hence we would expect infants and young children to be hypermetropic. This is found to be the case. Observations made on large numbers of cases have shown that in early infancy there is *always* hypermetropia to the extent of 2 to 5 dioptres. (A dioptre (D) is the strength of a lens of one metre focal length; 2 D would correspond to a lens of $\frac{1}{2}$ metre (= 50 cm.) focal length; 3 D, to a lens $\frac{1}{3}$ metre (= 33 cm.) focal length; 5 D, to a lens $\frac{1}{5}$ metre (= 20 cm.) focal length, and so on, it being remembered that the shorter the focal length of a lens the greater is its curvature and hence also its refractive power.) As the child grows older the degree of its hypermetropia gradually diminishes, and at 14 years of age it still amounts to 0·75 D. Indeed, it can be shown that were it not for the greater curvature of the infantile lens (see p. 544), and possibly also of its cornea, the axial length of the baby's eye should make it hypermetropic to the extent of about 30 D instead of 2 to 5 D.

The Refraction of the Eye.—The degree of the power of the eye to refract parallel rays of light is called the *refraction of the eye*. If it is such as to bring the rays to a focus on the retina the refraction of the eye is normal; if not, then there is an *error of refraction.*

Visual Acuity.—To discover errors of refraction one uses the visual acuity test. The smaller the object that an eye can dis-

tinguish at any fixed distance, or the greater the distance at which an object of a given size can be distinguished, the greater is the acuity of vision of that eye. The testing of visual acuity is done by means of Snellen's test cards, consisting of rows of letters of various sizes. Above each row is a number stating the distance in feet or in metres at which an average eye can read the letters in it with ease. The top row letters are of such a size as to be easily read by a normal eye at 60 metres, and the bottom line should be readable at 6 metres. The child is seated with one eye covered, but not compressed, with the palm of the hand, at a distance of 6 metres from the card which is properly illuminated, and the letters which he can read are noted. Supposing he can read the bottom line, which a normal person should read at 6 metres, then the child's vision with the uncovered eye is normal and is recorded as $\frac{6}{6}$, i.e., he can read at 6 metres what a normal person can read at 6 metres.

If he cannot read lower than the line marked, say 18, then it means that he can only read at 6 metres what should be normally read at 18, and his visual acuity is recorded as $\frac{6}{18}$, and so on. The process is repeated with the other eye.

Every teacher ought to be able to estimate visual acuity, and if it is less than $\frac{6}{6}$ with each eye the child ought to be referred to an ophthalmologist.

The school ought to have a number of Snellen cards, each containing different arrangements of letters, and they should be kept under lock and key in the intervals between tests so as to obviate the possibility of the children becoming familiar with the lettering.

In the case of young children who are unable to read, pictures of different sizes, such as a table, chair, hat, animal, or other familiar objects, should take the place of the letters in the rows.

In the case of still smaller children, only objective tests, such as retinoscopy, can be used. But this can only be done by the ophthalmic specialist.

But whilst the Snellen card will detect the grosser varieties of visual defects, it fails to detect those degrees of hypermetropia which the children can easily overcome and mask by accommodation. That this is the case is proved by the fact that whilst careful testing of a large number of children, 4 to $7\frac{1}{2}$ years old, by means of the Snellen type has shown that some 86 per cent. had normal visual acuity, Bishop Harman found that when accommodation is paralysed by the instillation of hematropine

and cocaine, children of the same age-group showed no more than about 2 per cent. of emetropia! 65·6 per cent. of the children had hypermetropia up to 2 D or astigmatism up to 0·5 D, or both, i.e., degrees of error of refraction that would on account of the exercise of accommodation have normal visual acuity. To detect such smaller visual defects, one should test visual acuity with convex lenses in front of the eye. The lens which just fails to give $\frac{6}{6}$ roughly measures the degree of visual defect.

The following statistical table represents the results of the examination of 3,000 children, 8 to 12 years of age, in Liverpool:—

TABLE CLXXIV.

Vision	Number of children	Per cent.
Hypermetropia	1,085	36·0
,, with astigmatism	981	32·7
Myopia	276	9·2
,, with astigmatism	356	11·9
Mixed astigmatism	218	7·3
Emmetropia	61	2·0
Not possible to determine	23	0·8
	3,000	100·0

Prevention of Hypermetropia.—As hypermetropia is the normal condition of the infantile eye, it is clear that its persistence means a delay in its development. Unfortunately we are ignorant of the causes of such delay and are therefore powerless to prevent it, in the same way as we are unable to prevent congenital heart disease or the condition of undescended testicles. The same remark applies to astigmatism. It is important, however, that all such cases should be detected as early as possible, in order that the use of such eyes should be suitably graded and regulated and the defect remedied when necessary by suitable glasses. Theoretically all young children are in need of glasses for their hypermetropia, but, owing to the great range and freedom of their accommodation, the majority of cases can easily do without them. Indeed, it would be as great a mistake to fit all children with glasses and thus weaken their ciliary muscles by depriving their eyes of the exercise of accommodation, as it is to put all children into corsets to hold up their backs and thus weaken the spinal muscles. It is only when symptoms of eye-strain appear that their eyes require the artificial aid of glasses.

Myopia in Children.—No child is ever born myopic, but as it grows older the eyeball lengthens and its natural hypermetropia gradually diminishes. In some cases, e.g., after an attack of an infectious disease, the eyeball is soft and its tissues yield, so that the axial length increases more rapidly than normally, and the eye passes through emmetropia to myopia. Myopia may be of two kinds, viz., (1) stationary, or (2) progressive. In the stationary variety the myopia reaches a moderate degree and then stops. In the progressive variety, constituting about 10 per cent. of all cases of myopia, the myopia goes on steadily increasing and the globe becomes so distended that it becomes oval instead of spherical. In a case recorded by Donders the axial length reached 37 mm. instead of the normal 23. This type of myopia is a very serious one, since detachment of the retina or other grave conditions leading to total blindness of the eye may result. As it is not possible to tell at first to which type any given case of myopia belongs, all myopes must be dealt with seriously.

Causes of Myopia.—Before one can formulate definite rules for the prevention of myopia, we must clearly understand the causes which bring it about. These, however, are not known with any degree of certainty. We know that it is due to a gradual yielding of the coats of the eyeball, but when we come to consider why the coats should yield in some children and not in others, we begin to tread on debatable ground. Hermann Cohn, in 1866, published the results of examination of 10,000 children, in which he showed that the percentage of myopia increased from 1·4 in village schools to 59·5 in university students, as in the following table :—

TABLE CLXXV.

Class of School	Percentage of myopia
5 Village schools	1·4
20 Elementary schools	6·7
2 Higher girls' ,, ...	7·7
2 Intermediate ,, ...	10·3
2 Gymnasia	26·2
University	59·5

He also found that there is an increase in the percentage of myopia as one ascends from class to class in the various schools. He therefore concluded that myopia is originated and increased

by near work. This view was accepted by Virchow, Erismann, von Bolt and others, but the following facts militate against such a theory :—

(1) Examinations of military recruits show that high myopia (over 9 D) is commonest in classes who do not use their eyes for close work.

(2) Myopia is often monocular, the other eye being emmetropic. Hence it cannot be due to near work.

(3) Karl Pearson,[1] using Cohn's own statistics, finds that whilst the correlation coefficient between heredity and degree of myopia is between 0·60 and 0·90, that between the age at which the child begins to read and the degree of myopia is between − 0·08 and − 0·15, i.e., the later the child starts the school the worse the myopia. Pearson believes the correlation to be sensibly zero, so that age of starting has no relation to the degree of myopia.

The theory which prevails at the present time is that myopia occurs in children hereditarily predisposed to it, but is originated and increased by near work. The children of near-sighted parents show a greater predisposition to myopia than those whose parents have normal sight.

As to how near work produces near sight, there are many theories, for which books on ophthalmology should be consulted. **The prevention of myopia** lies in the following :—

(1) Remove all causes which produce weakening of the sclerotic, such as the infectious diseases and malnutrition, and no child recovering from an infectious disease should be allowed to strain his eyes. This applies especially to boarding schools, where, although the children are well looked after during an illness, they are left to do pretty much as they like, and read as much as they like, during convalescence.

(2) Strengthen the child's general constitution by plenty of outdoor life.

(3) Do not allow a young child to strain the eyes by using them for very small or very near objects, since this tends to elongate the eyeballs in order to get a good focus, and may result in myopia, especially in children hereditarily predisposed to myopia.

(4) Whilst ordinary children may start schooling at about 6 or 7 years of age, those that have an hereditary tendency to myopia should not begin school before the age of 12.

[1] *Brit. Med. Journ.*, July 17, 1909.

(5) Attention must be paid to (*a*) the lighting of the school rooms, (*b*) the school books, and (*c*) the school furniture.

(*a*) *School Rooms.*—Statistics show that visual defects are more common in badly-lighted and badly-ventilated schools. The window area should be 1 sq. ft. to every 5 sq. ft. of floor space. There should be no high buildings near the school to obstruct the admission of sunlight. The sitting should be so arranged that the light falls upon the pupils' desks from behind and the left. This will ensure that no light is thrown directly upon the child's face, or is directly reflected into his face. The light coming from the left will also obviate (in right-handed children) any shadow that may be cast by the hand or arm upon the writing paper.

(*b*) *School books* should have sufficiently large print to be seen distinctly a good distance away from the eye, and thus prevent the child from stooping. The height of the letters should be not less than 2·6 mm. for younger children and not less than 1·6 mm. for older. The letters should be properly spaced and should not be more than sixty in number, per line. The lines should not exceed 4 or $4\frac{1}{2}$ in. in length, so as not to fatigue the eye-muscles in consequence of undue rotation of the eyeballs. For the same reason the books must not be held too near the eyes. The lines should be at least $\frac{1}{10}$ in. apart. The paper should not be so highly glazed as to reflect the light into the eyes and thus cause glare. Half-tone illustrations should therefore be printed as separate plates. The print should be as black as possible, so as to give the greatest contrast with the white paper. Coloured inks are not seen so distinctly as black. Yellow letters in some school books were found to be invisible a foot off, even with glasses of + 2 D. Hence, to the large majority of young children whose hypermetropia is of that degree, these letters must be illegible.

(*c*) *School Furniture.*—The school walls should be painted a light buff colour. The wooden finish, as well as the desks, should be light. The blackboards should be dull black, so that no light is reflected from them.

(6) Regular eye-testing should be carried out, and any myopia detected should be forthwith remedied.

(7) When myopia has developed, it should be dealt with according to its degree: (*a*) Mild cases should, in addition to the precautions enumerated above, carried out with particular stringency, be fitted with glasses and be left in the ordinary school; (*b*) children with higher degrees of myopia (i.e., between − 5 and − 15 D) should be segregated in special myopia classes, established under the Elementary Education (Blind and Deaf) Act, 1893, so that they can receive instruction specially adapted to their needs; (*c*) the highest degrees of myopia (i.e., higher than − 15 D) must be sent to "blind schools."

(*c*) **Squint.**—Hypermetropia is often associated with internal squint of the worse eye. (For explanation, see "Child Physiology," p. 600.) This results in double vision, and the brain, in order to do away with such an annoyance, soon learns to disregard the image formed by the squinting eye. But if this continues for long, the constant refusal of the brain to receive visual impressions from the squinting eye makes the latter refuse to receive or send such impressions, and ultimately that eye becomes blind. Hence, all cases of squint (or strabismus) must be attended to at once—not only for æsthetic but for utilitarian reasons. The eyes should be fitted with spectacles as soon as squint is noticed, even in very young babies, unless the condition is very transitory. In addition to providing accurate spectacles, the squinting eye (or "lazy eye," as it is sometimes called) should be made to work by bandaging up the other eye for several hours a day. By such treatment the child loses the squint after a time (a few years), and the vision remains even in the squinting eye. If neglected, not only does the squinting eye become incurably blind, but the deformity can only be remedied by operation.

Temporary squinting in young babies may be disregarded. It is due to inability to co-ordinate the action of their ocular muscle and soon disappears. It is only when it persists that it requires treatment.

(*d*) **Injuries.**—Many eyes are lost annually as the result of hitting against pointed instruments, such as pens, knives, pointed sticks, &c., either carried by the children themselves (when they stumble and fall forward upon the instrument), or carelessly carried by other people. No child should be allowed to play with pointed instruments, and the habit of carrying pens between the ear and the side of the head should be discontinued.

Other dangerous practices are playing with catapults and air-guns. The latter especially are very dangerous weapons. The force of the air driven out of the gun, even unloaded, is enough to burst the eye on looking down the gun when firing it off.

The above remarks apply to all children, but especially to children who for some reason or another are blind in one eye. Such children should not be allowed to run the risk of losing the only useful eye they have by taking part in such risky sports as rackets, golf, or boxing. The racket ball is so small that it may enter the orbit and burst the eye almost to shreds. Golf balls are also small enough to go a certain way into the orbit, and if they come with great force may inflict sufficient injury to render the eye blind. Tennis and cricket balls, being large, are stopped by the bony boundaries of the orbit, and the eye which quickly recedes into the orbit escapes with perhaps some slight superficial injury. This, of course, is truer still in the case of footballs. In the case of boxing, although the risk is exceedingly small with closed gloves, it is considerable if an accidental blow is received from the point of an open thumb.

Staring at the sun, during eclipses, with eyes unprotected by smoked glass or blackened photographic films, is followed by blindness in a large number of cases. It is no protection to look at the reflection of the sun in a pail of water, as is often ignorantly done. Even when suitably protected, one should not look at the eclipse for more than a few seconds at a time.

To look at the sun not during an eclipse, as is often attempted by foolish boys out of a spirit of bravado, is, of course, still more dangerous.

The other sense organs are the nose, skin, and tongue. These are referred to in other parts of the book.

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PART III

POST-NATAL HYGIENE

SECTION III

CHILDHOOD STAGE

"It may be doubted whether the pleasure of seeing children ripened into strength be not overbalanced by the pain of seeing some fail in the blossom and others blasted in their growth."—Dr. S. Johnson.

CHAPTER XXIV

THE PREVENTION OF INFECTIOUS DISEASE

"... To take arms against a sea of troubles
And by opposing end them."

SHAKESPEARE.

THE principal infectious diseases occurring in children of various ages are; Ophthalmia neonatorum, summer diarrhœa, measles, whooping-cough and diphtheria, with their complications (especially broncho-pneumonia), chicken-pox, mumps, scarlet fever and tuberculosis. Small-pox is now, thanks to Jenner's discovery of vaccination, practically an unknown disease.

Each period of infancy and childhood has its own susceptibility to particular diseases. Ophthalmia neonatorum, as its name implies, attacks babies during the first few days of life; summer diarrhœa claims, as its victims, babies during early infancy; whilst the ordinary fevers, measles, whooping-cough, scarlet fever and diphtheria, although they may affect very young infants, generally attack children during later infancy and childhood. The slight susceptibility of very young infants to these diseases is, as we have already explained in a previous chapter (p. 266), due to the presence within their systems of immune bodies that had been transferred to them from mothers, previously affected with these diseases, through the placenta. For a similar reason, i.e., on account of the presence of these immune bodies in the mother's milk, breast-fed babies are less liable to these diseases than are artificially-fed babies (see p. 152).

The period, 1 to 5 years, is one in which children are not only very liable to get the various infectious diseases, but is the one in which the fatality from these diseases is highest. The following statistics relating to measles will illustrate this point. Out of 3,181 cases of measles treated at the Glasgow Fever Hospital during 1897-99, the case mortality at all ages was 9.68 per cent. distributed as follows :—

Table CLXXVI.

Ages	0-1	1-2	2-3	3-4	4-5	5-10	10-15
Mortality per cent. ..	23·4	32·6	17·6	9·8	3·7	3·2	3·5

These figures show that the maximum death-rate is at the age 1 to 2 years. Figures show that 90 per cent. of the deaths from measles occur amongst children under 5 years of age; 98 per cent. in children under 10, and the remainder after the age of 10. For these reasons it is obvious that even if we cannot entirely abolish all the infectious diseases, we can win the largest part of the battle if we stave off these diseases to as late an age as possible.

It is important to bear this fact in mind in order not to acquiesce in the very popular error that, "As every child must at one time or another suffer from some such disease as measles, whooping-cough, or scarlet fever, &c., the sooner the child gets it the better it is." Acting upon this belief, it is the practice amongst the less enlightened to expose every young child to any case of such disease that may occur in the house in order that it may get the disease and be done with it. This is a silly and dangerous practice, because, as we have seen, the younger the child the greatest is the danger. Moreover, it is not absolutely necessary for a child to have such diseases at all. It is true that on account of the extreme infectivity of these diseases the largest majority of children get them at some time or another, but there are many people who have never suffered from these diseases at all (see below, p. 571). Another fact to remember about these diseases is, that even if young children recover from these diseases they may remain affected with certain complications which may invalid them or cripple them for life; e.g., tuberculosis and chronic bronchitis, &c., may follow measles and whooping-cough. These complications are less liable to occur in older children.

The reason why infectious diseases occur as a rule during childhood is twofold: (1) During infancy, there is in most babies a passive immunity derived from the placental circulation as well as perhaps from the breast milk. (2) In adult life, there is generally an active immunity derived from an attack of the disease in childhood. Indeed, when any such disease is introduced into a population that had never been visited by the disease before, it attacks young and old alike. Thus, in 1846 an epidemic of measles occurred for the first time in the Faroe Islands, when 6,000 out of a population of some 7,800 were attacked.

Cause of Infectious Disease.—There is no doubt whatever that all infectious diseases are due to micro-organisms which have settled and multiplied in the body, and that each disease is produced by its own specific organism. In the case of some of these diseases the organisms have been actually identified and isolated (e.g., tuberculosis, diphtheria, typhoid fever, ophthalmia neonatorum); in the case of some of the others (e.g., measles, German measles, small-pox, chicken-pox, &c.) the causative organisms have so far eluded discovery, probably because the organisms responsible for these are of the nature of ultra-microscopic viruses. In the case of others again, like whooping-cough and scarlet fever, certain bacteria have been described as being the specific organisms of these diseases, but their identity has not yet been definitely established.

The development of an infectious disease may be compared to the growth of a plant. Just as in the case of a plant, we must not only sow the seed, but the soil must be put under favourable conditions of moisture, temperature and light, so in the case of infectious disease we must have not only the seed, i.e., the microbe, but the soil, i.e., the child's body must be in such a condition that the microbe can grow and thrive on it. It is for this reason that a child that is in a good state of health and nutrition does not so easily get an infectious disease as one that is in poor health, e.g., suffering from rickets, anæmia, &c.

Again, just as in the case of plants, each plant has its own particular seed, so that an apple tree, for example, will grow from apple seeds only, and vice versa, so in the case of infectious diseases each disease is caused by its own particular organism and no other. In other words, infectious diseases breed true.

To pursue the analogy still further. When seeds are put into the soil the plant does not begin to grow at once—it takes some time for the seed to develop; so when microbes invade the body they do not give rise to the disease immediately, but there is an interval of time during which the microbes develop and multiply, and give rise to poisons or toxins whose entry into the blood produces the symptoms of the disease. This interval, from the moment the microbes have reached the body until the disease has shown itself, is called the *incubation period* of the disease. During this period the patient is, as a rule, quite ignorant of the fact that infection has occurred.

Each infectious disease has its own incubation period, as follows:—

Scarlet fever	2-7 days	
Measles	10-14 ,,	
German measles	8-21 ,,	
Whooping-cough	7-14 ,,	
Diphtheria	2-8 ,,	(but not known with certainty)
Chicken-pox	10-14 ,,	
Mumps	10-21 ,,	
Small-pox	9-15 ,,	
Typhoid	7-21 ,,	

The Conditions Favourable for the Growth of Bacteria.—Bacteria grow best under conditions of warmth, moisture, darkness and suitable food, and when there are no inhibitory agencies at work, such as the presence of antagonising organisms, or of antibodies in the blood. In the case of pathogenic organisms entering the body through the digestive and respiratory tracts, decayed teeth, as well as diseased tonsils and adenoids, or other catarrhal conditions of the nose, provide the most suitable environment for their growth. Hence proper dental, oral and nasal hygiene forms a most valuable weapon in the prevention of infectious disease. The following *preventive rules* should therefore be observed by every child :—

(1) The mouth should be washed out and the throat gargled every morning and evening, either with ordinary water or preferably with some mild antiseptic solution, such as boracic acid or glycothymoline, &c.

(2) The teeth should be brushed night and morning.

(3) The teeth should be periodically examined by a dentist, and any carious teeth discovered should be remedied.

(4) The presence of septic tonsils and of adenoids that obstruct breathing should be remedied, either by medical or, if necessary, by surgical means.

(5) The nose should be blown into a clean handkerchief at regular periods, varying with each child. One suffering from slight nasal catarrh should blow the nose frequently during the day. The child must be taught the way to do this. The sides of the nose must be wrapped in the handkerchief, and whilst the mouth is closed and one nostril is shut by one finger, a forcible deep expiration is made through the other nostril until it is entirely emptied of mucus. The process is repeated with the opposite nostril. Such *nasal drill* followed by deep respiratory exercises (breathing with

the mouth closed) is of great value in the prevention and treatment of nasal discharges and adenoids.

(6) No child may lend his or her handkerchief to another.

(7) The habit of chewing pencils or penholders, or "giving others a bite," must be strictly avoided.

(8) The hands must be kept scrupulously clean, and should be thoroughly washed and soaped before meals.

(9) The bowels must be made to act every day.

(10) The child should be out in the open-air as much as possible.

Infection versus Immunity.—The relationship between infection and immunity can be considered in the same way as the well-known school problem about the time it would take to fill a tank of known capacity, C, if it is supplied with water at a certain rate, S, from a pipe at the top, and at the same time discharges water at a rate, D, from the bottom. It is obvious that in a unit of time the amount of water remaining in the tank would be $S-D$, and therefore the time it would take to fill the tank would be $\frac{C}{S-D}$.

If we call C the "critical dose," i.e., the minimum number of organisms necessary to produce infection, S the number of bacteria that the individual receives per hour, and D the number of organisms which the individual's defensive mechanism can destroy in an hour, then obviously it will take $\frac{C}{S-D}$ hours for the body to harbour a critical dose, C; i.e., for the disease to establish itself.

If D is either equal to or greater than S, i.e., if the body can destroy the organisms at the same rate as, or at a greater rate than, it receives them in any particular case, then the individual can be permanently exposed to the disease without the risk of contracting it. Indeed, his immunity will be gradually increased, so that he will be able to withstand more massive doses.[1]

The value of D naturally varies with the individual. Some people are more immune than others. The value of S varies with the nature of the disease, the distance between the individual and the source of infection, and a number of other

[1] See Sheldon F. Dudley, "Fundamental Factors concerning the Spread of Infectious Diseases." *Lancet*, June 7, 1924

factors. Thus, when a patient coughs the droplets can carry 30 to 40 ft.; beyond that distance there is practically no risk of infection. But if the distance is halved, then, by the familiar law of inverse squares, the number of droplets reaching the individual is increased four times. Vice versa, if the distance between the source of infection and the individual is doubled, the chance of infection is reduced four times.

Similarly, the virulence of the source of infection would necessarily determine not only the number of organisms it emits, but also the size of the critical dose; and so also would the state of the intervening atmosphere, whether still or in motion, hot or cold, dry or damp.

If the air is in motion it will scatter and diminish S. If the air is cool and dry, droplets evaporate and are scattered, and so on. Hence good ventilation may so scatter the dose as to render it practically harmless.

From what has been said regarding the seed and the soil, we see that for the development of any infectious disease two things are necessary: (1) The presence within the body of the particular micro-organism, (2) the predisposition of the body to the growth of the organism. Hence it is possible for persons to carry disease organisms in their bodies without themselves being ill, because for some reason or another their tissues may not be predisposed to the organism's invasion. Such individuals are called **carriers**, and are a frequent danger to others whose resistance to the organism may be feeble. For example, many persons harbour diphtheria bacilli in their throats without themselves being affected, and if they convey them to others by kissing, coughing, or sneezing, &c., may infect those others with the disease, which may even turn out to be of a virulent character. They do not, however, always convey infection. Indeed, it is possible that by distributing small doses, which are not sufficiently massive to produce the disease, they may actually help to immunize those with whom they frequently associate (see p. 571).

Although the discovery of the microbic origin of infectious disease dates from the time of Pasteur, reference to certain minute invisible bodies in the air as the cause of disease is found in the ancient literature. Varro (116-27 B.C.), quoted by Hektoen makes the following statement: "Certain minute organisms develop which the eyes cannot see and which (being disseminated) in the air enter the body by way of the mouth and nostrils and give rise to serious ailments." A Talmudic Rabbi is also responsible for the remark that, "None could stand the sprites that fill the air, could the human eye but see them there."

A.—The Transmission of Infection from Person to Person.—*The body gives off infective material* in the following ways :—

(1) *The Skin.*—It is possible that the desquamation in cases of scarlet fever is infectious, but as the specific organism of this disease has not so far been isolated with certainty, it is not possible to say to what extent the infection is transmitted in this way. It is probable, however, that the infectivity of the skin is due to its contamination with excretory discharges.

(2) *The Alimentary Tract.*—Many diseases are conveyed by means of the fæces, e.g., typhoid fever and epidemic diarrhœa (soiled napkins), as well as broncho-pneumonia in babies, a disease in which Ribadeau-Dumas has recently shown that the organism is swallowed and eliminated through the bowel.

(3) *The Respiratory Tract.*—The sputum, either when expectorated or when sprayed in the course of conversation, &c., may convey the germs in cases of tuberculosis, scarlet fever, diphtheria, whooping-cough, measles, mumps and influenza. The nasal discharge, especially in measles and in common colds, is very infectious. Dudley[1] found that droplets in coughing can carry 30 to 40 ft., but that no infection occurs in wards full of coughing children if the beds are no more than 9 to 12 ft. apart, because the infection is received in low concentration at these distances.

(4) *The Urine.*—This carries the organism in cases of typhoid fever.

(5) *Other discharges*, e.g., the tears, or other discharges from the eyes as well as from the nose in the case of measles or ophthalmia neonatorum.

(6) *Intermediate Hosts.*—Infection is frequently conveyed from person to person through the intermediate agency of other animals, especially the fly or other insects.

(7) *The air* is negligible as a medium of infection. It has been found, for instance, that sewer air when free from splashing is practically free from pathogenic organisms, and surgeons have long given up the use of the carbolic spray introduced by Lister for disinfecting the air in the operating area.

B.—The Channels of Infection.—The body *receives* infection in the following ways :—

(1) *The Skin.*—So long as the skin is intact, it forms a successful barrier against the penetration of microbes, but the

[1] *Lancet.*, 1924, i.

slightest abrasion or other wound in the skin opens the door for them. Tetanus and erysipelas, for instance, start from the contamination of a superficial wound by the appropriate organism, and malaria is conveyed by the puncture made in the skin by a mosquito carrying the malarial organism.

(2) *The Alimentary Tract.*—Some germs reach the body by means of the food or water that is swallowed. This is the case with epidemic diarrhœa, typhoid, &c. Fortunately, in this country, food and water are not so contaminated as in other countries, especially in the tropics, and hence a disease like typhoid is now practically non-existent in England. But contamination of food, and especially of milk, still occurs through the agency of the common fly as well as in various other ways in the course of its transmission from the cow to the baby (see pp. 407 *et seq.*)

FIG. 144.—The House Fly: (*a*) Legs covered with hairs to which filth clings. (*b*) Glue pads on the feet on which stick numerous bacteria. (*c*) Trunk through which the fly sucks up filth and vomits it back on food.

The house-fly (fig. 144), in common with all other insects, passes through the ordinary metamorphoses of egg (ovum), maggot (larva), chrysalis (pupa) and the adult fly. The egg is laid in human or animal excreta, in the warmth of which it develops into the maggot and later into the chrysalis and perfect fly. When it is fully developed it feeds alternately upon excreta and human food (fig. 32, p. 154). Upon the former, which contains predigested food, it depends for its nitrogenous material, since the fly has no gastric or other proteolytic glands; whilst its non-nitrogenous food it obtains by attacking human food. It is this constant alternation between excreta and human food

that results in the contamination of the latter. The contamination occurs not only through the fly's feet, whose hair, claws and adhesive pads harbour numerous pathogenic organisms, but also through its proboscis, or elongated tube, through which it sucks up its food as well as its own excreta (fig. 144). Typhoid germs have been known to cling to a fly's foot for twenty-five days, and tubercle bacilli have also been found attached to the flies in the sick room of consumptive patients. Summer diarrhœa is, however, the chief disease (which concerns us in this book) that is conveyed through the milk or other food substances through the agency of the fly. It has been estimated that one grain of fæcal matter may contain as many as 16,000,000 infective organisms. If, in addition, we realise that each female fly lays fifteen eggs every day, that the complete hatching period of each egg is not more than a fortnight, and that the normal duration of life of a fly is eight weeks, it will be readily seen, even by the non-mathematical reader, that the number of flies hatched in a season out of each original fly is enormously large.

(3) *Respiratory Tract.*—Many diseases are acquired by breathing in organisms from the air, e.g., diphtheria, influenza, tuberculosis, the virus of poliomyelitis (infantile paralysis), measles, scarlet fever, small-pox, pneumonia, &c.

(4) *Placenta.*—The transmission of toxins and microbes through the placenta has already been considered (see Chapter VIII).

There are other channels of infection, e.g., *genito-urinary* tract in venereal disease, *auto-infection*, i.e., infection by an organism from another part of the patient's own body, e.g., *coli* cystitis or pyelitis, that is to say, inflammation of the bladder or kidney caused by the *Bacillus coli* which has penetrated from the bowel, where it is a harmless inhabitant, into the bladder, where it causes mischief.

The Control of Infectious Diseases.—From what we stated before it will be clear that there are two main methods to be adopted for the control of infectious diseases: (1) Those directed towards preventing the organisms reaching the body, i.e., diminishing the size of the invading dose, S (p. 563); and (2) those directed towards increasing the resisting power of the individual exposed to infection, so as to render him or her unfavourable soil for the organism to grow and multiply, i.e., increasing the value of D (p. 563) by increasing the defensive powers of the body, i.e., by producing immunity.

(1) Methods directed towards Preventing the Organisms from reaching the Body.

(*a*) *Notification.*—It is obvious that before a public health authority can take measures for stopping the spread of an infectious disease, it must first be made aware of its presence.

FIG. 145.—Children nursed with a case of scarlet fever in the same ward at Dr. Barnardo's Village Home Hospital. No cross-infection resulted.

The Infectious Disease (Notification) Act was passed in 1889. This Act, which though compulsory in London was "adoptive" for other districts, became compulsory for the whole of England and Wales in 1899. The diseases scheduled in the 1889 Act were small-pox, cholera, diphtheria, erysipelas, scarlet fever, typhus, typhoid, continued fever, relapsing fever and puerperal fever, and the local authority had the power to add to this number, e.g., chicken-pox, measles, &c., have from time to time been made notifiable by various local authorities. The duty of notifying these diseases was imposed upon the doctor or nurse in attendance upon the case, as well as upon the patient's nearest relative or other occupant of the house who happened to be aware of the illness.

(*b*) *Isolation.*—When the local authority is notified of the presence of any infectious disease, the medical officer of health for the district may either direct that the patient be removed to a fever hospital, or order that the patient be properly isolated at home. The strict isolation of scarlet-fever cases has been shown by the experience of the Barnardo Homes, where the Milne method is adopted, to be unnecessary. The Milne method consists in anointing the patient with eucalyptus oil (see fig. 145).

(*c*) *Quarantine.*—The medical officer of health will also order that no children that have come into contact with the patient be allowed to attend school until the quarantine period of the disease, which is usually taken as the longest possible incubation period together with an extra three days, have passed. By that time it will become impossible for the "contact" to convey infection to others.

The term quarantine is derived from "quaranta," which means 40, and was formerly applied to the custom of preventing passengers landing from a ship on which a case of infectious disease had occurred during a voyage until a period of 40 days had elapsed from the day of entering the harbour.

(*d*) *Disinfection.*—When the patient has been removed from the house, or has recovered from his illness (if he was not removed), the sick room is disinfected in one of several ways, e.g. :-

(i) *Burning Sulphur.*—2 lb. of sulphur for every 1,000 cubic feet of space are burnt in the middle of the room, after the windows, fire-places, doors, &c., are thoroughly pasted up. The room is then kept sealed up for at least twelve hours.

(ii) *Spraying with a Disinfectant.*—A 2 per cent. solution of formalin or a 1 per cent. solution of perchloride of mercury is sprayed in the room.

In addition, all articles likely to retain infection are either destroyed, e.g., books, cheap toys, &c., or, in the case of more costly ones, such as bedding, clothes, &c., disinfected by steam by the public health authorities.

If the patient is isolated at home, the urine and motions must be disinfected by mixing with 5 per cent. carbolic or 5 per cent. perchloride of mercury before being emptied into the w.c. Also all nasal discharges as well as sputa must be received into Japanese paper handkerchiefs and burned.

(*e*) *Destruction of intermediate hosts*, e.g., lice in typhus, rats in plague, flies in typhoid fever, epidemic diarrhœa, tuberculosis, &c., the common flea and bed-bug in cases of scarlet fever, &c. Lice can be got rid of by putting clothes either into a steam disinfector, or into a small air-tight closet in which a couple of pounds of sulphur is burned. Bugs are destroyed by stripping the wall-paper off and disinfecting by burning sulphur in the proportion of 5 lb. for every 1,000 cubic feet, i.e., two and a half times stronger concentration than is required for disinfecting a room. Flies can only be got rid of by doing away with manure and all kinds of refuse near the house where they can breed. Fly-papers and traps are also useful.

(2) **Production of Immunity.**—We have seen that when microbes invade the body and find a suitable soil upon it, they begin to germinate and multiply. In the course of this growth they manufacture poisons or toxins, the absorption of which into the blood gives rise to the symptoms of the disease of which they are the specific organisms. At the same time, as the poisons are being manufactured and poured into the blood, there is also an attempt made by the various constituents of the blood to fight against the organisms and their products. Some of the white cells of the blood assume the rôle of *phagocytes* (or eating cells) by attacking and engulfing the invading microbes. This phenomenon of *phagocytosis* was first discovered by Elie Metchnikoff, in 1884. Metchnikoff also discovered that when germs are introduced into any part of the body these phagocytes are attracted to that part—as the result, probably, of some chemical substance in the microbes having some affinity for the phagocytes. This attraction is spoken of as *positive chemotaxis.*

Sir Almroth Wright and his school have shown that before bacteria can be engulfed by phagocytes, they must first be acted upon by certain substances, called *opsonins*, in the blood.

In addition to the phagocytosis, the toxins are combated by the action of *antitoxins* in the blood. These antitoxins ultimately bring about the patient's recovery, and so long as they remain in the body they keep the latter in a state of protection or immunity against further attacks of the same disease.

It is to be remarked, however, that although there are many facts, viz., the various phenomena in connection with inoculation of toxins, which tend to support this *retention* theory of immunity (i.e., the theory that organisms growing in the body produce substances which tend to inhibit their growth), there are certain facts which militate against its validity. In the first instance, not only is it difficult to understand how these antitoxins can be retained for a long time in the body, but observation shows that they are in fact rapidly eliminated.[1] Further, the tissue fluids of protected animals form, as a rule, good culture media for the organisms that caused the disease.

Kinds of Immunity.—There are two kinds of immunity, viz.: (1) *Natural* or *innate*, and (2) *acquired* or *artificially induced.*

(1) *Natural Immunity.*—Some people or races, or animals, are immune to certain diseases. Thus, negroes are insusceptible to yellow fever, and the mongolians are said to be immune against scarlet fever. Human beings are also immune to cattle plague and other animal diseases, whilst animals are insusceptible to measles, whooping-cough, &c. Again, the Schick and Dick reactions which test the susceptibility of individuals to diphtheria and scarlet fever respectively (see pp. 577 and 584) show that some people are immune to these diseases. This immunity, however, is probably acquired rather than natural (see below).

(2) *Acquired Immunity.*—This may be of two varieties :—

(*a*) Active, i.e., immunity produced in an individual as the result of the manufacture of antibodies in his own blood consequent upon :—

(i) A past attack of the disease. This, of course, does not concern us here.

(ii) Frequent exposure to small doses of the organism, which may not be sufficient to produce the disease, but which suffice so to habituate the body cells as to enable them to protect themselves against these infections. Thus, during

[1] See W. M. Feldman's "Biomathematics," London, 1923, p. 218.

an epidemic of diphtheria in the Greenwich Naval School, many Schick-positive (and therefore susceptible, see p. 577) boys became Schick-negative (and therefore immune) without having shown any signs of diphtheria, and without having received any prophylactic inoculation. These boys have, therefore, become immune by auto-inoculation—the so-called "herd" immunity. This phenomenon to some extent explains the comparative immunity enjoyed by doctors and nurses in fever hospitals, as well as the greater immunity of urban—as compared with rural—populations to infectious diseases, because the former are repeatedly exposed to small doses of infection. Hence, it is probable, as remarked above, that the immunity of certain individuals to certain infections is acquired rather than natural. In the Greenwich Naval School the Schick-positive boys who became Schick-negative during an epidemic proved, on bacteriological examination, to be carriers, and when a number of new Schick-positive boys were introduced into the school, 50 per cent. of them became Schick-negative without having shown signs of diphtheria. Hence carriers may possibly protect as many as they infect.[1] (See above, p. 564.)

(iii) Inoculation or vaccination with the virus or organism, or its products, modified or attenuated by :—

(α) Passage through lower animals (e.g., vaccination against small-pox).

(β) Other means, e.g., vaccination against tuberculosis (see p. 616).

(*b*) Passive, by which is meant the immunity brought about in the individual by the introduction into his blood of immune bodies obtained from the blood of other individuals (human or animal) that had passed through an attack of the disease, e.g., antitoxic serum

[1] See M. Greenwood, E. M. Newbold, W. W. C. Topley, and J. Wilson, "On the Mechanism by which Protection against Infection in Diseases is Acquired in Natural Epidemics," *Journn. Hyg.*, xxv, 1926, p. 336.

for diphtheria. The temporary immunity of infants who acquired it from their mothers, either through the placental circulation or from the milk, belongs to this type. The immunity conferred by serums (passive immunisation) is more rapidly produced, since it saves the patient the time necessary to manufacture his own antibodies; on the other hand, it is of much shorter duration, since the protective substances or antibodies so introduced are much more quickly eliminated from the body. For these reasons passive immunity by means of serum is indicated when the danger of infection is very close at hand (e.g., in cases of diphtheria), or when vaccines cannot be prepared (e.g., measles, &c.).

It is important to bear in mind that, although the above are the general methods of combating infectious diseases, there are no two diseases which can be successfully dealt with from the preventive point of view by identical methods. Some are best dealt with by one or other of the methods described for preventing the infective material reaching the body, whilst others are most successfully prevented by the methods of immunisation outlined above.

Hospital isolation, for instance, is—except perhaps in the case of small-pox—of very little value in stopping the spread of infection. Thus, the incidence of diphtheria has not diminished, in spite of the strictest isolation carried out for a large number of years. Terminal disinfection again has not diminished the number of cases of infectious disease, as the following statistics show in the case of diphtheria and scarlet fever:—

TABLE CLXXVII.

Years	Disease	Infected families	State with regard to terminal disinfection	Recurrences	Ratio
1902-05 ..	Diphtheria	1,457	Yes	25	1·71
1905-12 ..	,,	3,658	No	64	1·75
1903-09 ..	Scarlet fever	2,428	Yes	36	1·48
1908-12 ..	,,	1,704	No	26	1·53

In certain parts of England, for example Brighton, terminal disinfection after diphtheria and scarlet fever has been given up since 1910, and no increase in the number of recurrences of the diseases in the families has occurred. Sunlight (especially the

ultra-violet rays) and fresh moving air form very efficient germicidal agents for most organisms. Unfortunately, the amount of sunshine in most places in this country is so variable, and the amount of ultra-violet radiation that filters through our atmosphere is so uncertain, that in most cases we cannot safely rely upon this method of disinfection.

Diphtheria.

Mortality.—The following statistics relating to diphtheria are instructive :—

TABLE CLXXVIII.

Age-group	Morbidity (case) rate per 100,000		Percentage distribution of cases		Mortality (death) rate		Percentage distribution of deaths		Proportionate mortality per cent.		Fatality per cent.	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
0-1	629	371	4·4	2·0	90	278	7·7	20·0	0·7	2·8	14	75
1-4	1,340	1,630	35·0	33 9	96	146	30·7	40·0	10·5	15·0	7	9
5-9	1,262	1,600	39·3	40·4	100	100	38·5	33·3	38·4	38·4	8	6
10-14	334	528	9·4	12·1	22	22	7·7	6·7	14·3	16·7	7	4
15-19	153	82	4·4	2·0	22	—	7·7	—	4·5	—	14	—
20-29	76	106	5·0	6·1	9	—	7·7	—	2·2	—	13	—
30-39	48	55	2·5	2·5	—	—	—	—	—	—	—	—
40-49	—	14	—	0·5	—	—	—	—	—	—	—	—
50-59	—	—	—	—	—	—	—	—	—	—	—	—
Total	308	349	100·0	100·0	25	26	100·0	100·0	1·8	2·0	8	7

These statistics for Cambridge, Massachusetts, in 1915, show that the minimum incidence with the highest fatality-rate occurs during the first year; the maximum age incidence occurs during the age-period 1 to 10, viz., the school age. The percentage distribution of cases is about the same for males and females. As regards the fatality-rate, the difference between the rates in males and females is to be accounted for by the small numbers upon which these statistics are founded. Thus, during the age-period 15 to 19, there were seven cases of diphtheria in males, with one death, giving a fatality-rate of 14 per cent., and four cases in females, with no deaths, giving a fatality-rate of 0 per cent. J. D. Rolleston, out of 3,000 cases, finds that twenty-three occurred during the first year of life, and out of these ten died—fatality-rate = 43·9 per cent., whilst the total number of deaths was 234, giving a fatality-rate of 7·8 per cent.

Dr. C. V. Chapin, of Providence, R.I., collected statistics of

12,936 cases of diphtheria at all ages between 1889 and 1915. The following are his results:—

TABLE CLXXIX.

Age	1889-1914			1915		
	Cases	Deaths	Fatality	Cases	Deaths	Fatality
0-1	280	96	34·28	21	2	9·52
1	706	247	34·99	43	6	13·95
2-4	3,322	697	20·98	181	24	13·26
5-9	4,541	460	10·13	219	15	6·85
10-14	1,801	83	4·61	79	3	3·79
15-19	616	26	4·22	20	1	5·00
20	1,670	40	2·39	62	2	3·23
Total	12,936	1,649	12·74	625	53	8·48

The fatality-rate (i.e., case mortality) as well as the mortality (per 100,000 living persons) has been gradually declining. There seems to be very little doubt but that this decline is to a very large extent due to the use of antitoxin, although to some extent the decreased fatality-rate is quite certainly due to improved diagnosis based on bacteriological methods, which has been responsible for the detection of a large number of mild cases that had hitherto remained undiagnosed.

TABLE CLXXX.

Period	Antitoxin used			Antitoxin not used		
	Cases	Deaths	Fatality	Cases	Deaths	Fatality
1895-99	3,296	357	10·8	2,444	528	21·6
1900-04	4,970	365	7·3	1,289	256	19·4
1905-09	5,348	331	6·2	622	144	23·2
1910-14	6,409	379	5·9	505	89	17·6
1915	1,085	48	4·4	22	4	18·1
Total	21,108	1,480	7·0	4,882	1,021	20·9

The fact that the mortality-rate (*per* 100,000 *living persons*) has declined in twenty countries from about 80 before the antitoxin period to 16 since the introduction of antitoxin (i.e., a decrease of 80 per cent.) offers better evidence in favour of antitoxin, since in serious cases the question of inexact diagnosis before the antitoxin period could hardly arise. But such

evidence is not conclusive, since the objection can be raised that the decline is due not to the effects of antitoxin but to a milder type of the disease. The following statistics, which compare the fatality-rate of cases of diphtheria in the same town (Newark, N.J.) during the same periods, amongst patients who received antitoxin, with those who have not received it, affords the most conclusive direct evidence of the value of the serum. (See Table CLXXX.)

Further evidence is afforded by the fact that whilst in the pre-antitoxin period the fatality-rate (i.e., case mortality) was almost the same whether the patient came under treatment on the first, second, third, or subsequent days of illness, statistics published by the Metropolitan Asylums Board show that with the administration of antitoxin the earlier the injections are given the less the fatality-rate. Thus :—

TABLE CLXXXI.

Day of disease on which case first came under treatment	1894 Pre-antitoxin period	1896 Antitoxin period	Difference
First	22·5	4·7	17·8
Second..	27·0	12·8	14·2
Third	29·4	17·7	11·7
Fourth	31·6	22·5	9·1
Fifth and over	30·8	24·6	6·2
All cases	29·6	20·8	8·8

Moreover, antitoxin treatment has reduced the fatality-rate of all laryngeal diphtheria cases, where the question of mistaken diagnosis could hardly enter, from 62 per cent. to 11·7 per cent., whilst that of tracheotomy cases, where the possibility of mistaken diagnosis is altogether excluded, has been reduced by antitoxin from 70 per cent. to 20 per cent. It is estimated that no fewer than a quarter of a million diphtheria deaths are being prevented annually throughout the world as the result of antitoxin treatment.

Incidence.—Notwithstanding the use of antitoxin in reducing the fatality-rate of diphtheria, the *incidence* of the disease has, in spite of hospital isolation, quarantine and terminal disinfection, remained practically the same, and diphtheria is still responsible for some 5,000 deaths per annum in England and Wales, half of which occur under 5 years of age, as the following table shows:—

TABLE CLXXXII.—MORTALITY FROM DIPHTHERIA IN ENGLAND AND WALES, 1909-1913.

Age at death	Number of deaths		Rate per 100,000		Percentage distribution of deaths by age	
	M.	F.	M.	F.	M.	F.
0-1	632	468	31·9	24·1	5·5	3·9
1 year	1,417	1,259	75·6	68·1	12·3	10·4
2 years..	1,474	1,328	74·2	67·3	12·8	10·9
3 ,, ...	1,679	1,607	86·2	82·5	14·6	13·2
4 ,, ...	1,582	1,627	82·6	85·2	13·8	13·4
Under 5 years	6,782	6,289	69·9	65·4	59·0	57 8
5-9 years	3,736	4,740	40·3	57·1	32·5	29·0
10-14 ,, ...	621	705	7·1	8·0	5·4	5·8
15-19 ,, ...	134	122	1·6	1·4	1·2	1·0
20 + years	230	296	0·4	0·5	1·9	2·4
All ages	18,287	18,441	13·2	13·0	100·0	100·0

The introduction of the Schick test, together with the immunisation (as worked out by Park and his co-workers) of all those whom the test shows to be susceptible, enables one to reduce the incidence of the disease almost to zero.

The Schick Test for the Susceptibility of an Individual to Diphtheria.—This test, which was discovered by Bela Schick, of Vienna, in 1913, consists in the injection of $\frac{1}{50}$ of a minimal lethal dose[1] ($\frac{1}{50}$ M.L.D.) of diphtheria toxin intradermally. A positive reaction, shown by redness of the skin near the puncture, followed by pigmentation and desquamation, means that the individual is susceptible to diphtheria. A negative reaction, indicated by the absence of these phenomena, shows that there is present in that individual an amount of antitoxin equivalent to $\frac{3}{100}$ of a unit per cubic centimetre of the patient's serum, which is enough to confer immunity. It means, therefore, that the individual is not susceptible to the disease, and may safely be allowed to mix with diphtheria cases. The redness appears within twenty-four hours in 95 per cent. of cases.

The Park Modification of the Schick Test.—Park finds that a subcutaneous injection of 1 c.c. of the standard toxin-antitoxin solution into the anterior part of the forearm gives the same reaction as Schick's test. The advantage of this modification is that immunisation is carried out at the same time and thus

[1] A minimal lethal dose (M.L.D.) is the amount of toxin necessary to kill a guinea-pig weighing 250 to 300 grm.

saves time and expense. If there is no reaction the person is immune and no further treatment is necessary. If any reaction occurs, he receives two more injections at intervals.

Until the introduction of the Schick test, the only method of detecting susceptible cases was the complicated one of examining specimens of the person's blood for antitoxin. The Schick test has placed in the hands of the medical practitioner a simple, quick, safe and absolutely reliable method of diagnosing the susceptibility of a person to diphtheria.

Acquired Immunity from Frequent Exposure to Mild Doses.—As we shall see (p. 585), children of the better social classes, and who are therefore comparatively more isolated, show a smaller percentage of immunity, as tested by the Schick reaction, than those belonging to a lower social class. (See also p. 572.)

Artificial Immunisation.

In 1903 Theobald Smith suggested the use of *toxin-antitoxin* mixtures for the purpose of immunisation against diphtheria, and in 1913 von Behring first utilised this suggestion. The first to carry out such immunisation on a large scale were Park and Zingher, in the United States, in 1913. The method originally used was to give three weekly injections of 1 c.c. of the toxin-antitoxin mixture, having the following composition: 3 L + doses of toxin, and 3·5 units of antitoxin. One preferably uses now toxoid-antitoxin, toxoid being diphtheria toxin treated with formalin, and a $\frac{1}{10}$ L + dose can be used instead of 3 L +. This dose is said to give the same degree of immunity without any local reaction. (An L + dose of toxin is the amount which mixed with 1 unit antitoxin kills a 250 grm. guinea-pig in four days.) These injections may be given to all young children without subjecting them to the Schick test, since, as we have seen, the proportion of susceptibles is so high amongst them that it is unnecessary to go to the trouble of testing. Moreover, in very young infants a negative Schick test is probably due to immunity transferred from the maternal circulation, which is very transient. In older children (e.g., in schools, &c.), those that give a positive reaction with the Schick test should be immunised. Those that are negative are safe against infection. Experience has shown that three injections will produce active immunity in 60 to 80 per cent. of the susceptible cases, and a further course of injections will successfully immunise the remainder.

The following statistics, given by Ker and McGarrity, are from the Edinburgh City Hospital. They show that under six months the large majority of infants are insusceptible (on account of their having absorbed antitoxin through the placental circulation), and that the age-period 1 to 2 years is the one in which there is the highest susceptibility (81 per cent.). It will also be seen that 75 per cent. of children under 5 are susceptible:—

TABLE CLXXXIII.

Age		Number tested	Per cent. positive (i.e., susceptible)	
0-5	0-½ year	13	15·4	75 per cent.
	½-1 ,, ...	40	75·0	
	1-2 years	105	80·9	
	2-4 ,, ...	313	77·0	
	4-5 ,, ...	124	69·3	
	5-10 ,, ...	539	58·4	
	10-15 ,, ...	250	52·0	

It has been found that the babies of Schick-positive mothers are themselves Schick-positive, and vice versa, showing that the infant's immunity was transmitted to it from its mother. Similar statistics have been published by other observers in England, America, and other parts of the world.

The method of immunisation by toxin-antitoxin should be carried out in all schools, hospitals, crêches and other institutions where children are admitted, in the case of the Schick-negative inmates. It is probably no exaggeration to say that no child need nowadays die or suffer from diphtheria. Zingher, who immunised tens of thousands of cases, did not meet with a single accident.

The method is therefore perfectly safe (provided no frozen toxin-antitoxin is used, since freezing precipitates the toxin and separates it from the antitoxin),[1] it gives an immunity which lasts for a number of years (Zingher found that patients gave a negative Schick test eight years after immunisation), and **in view of the fact that 75 per cent. of children under 5 years old are susceptible to diphtheria, and that during this age-period the mortality is highest, all children should be immunised during this period, preferably at about 2 years of age.** There is very

[1] In the winter of 1923-24 some children in Massachusetts were injected with a toxin-antitoxin solution that had been frozen in the particularly cold weather. All the children became ill, although no fatalities occurred. No harm has ever resulted from solutions that had not been frozen (*Lancet*, 1924, i, 505).

little doubt that universal use of the Schick test, followed by immunisation by means of toxin-antitoxin, will stamp out diphtheria as surely as vaccination has wiped out small-pox.

The cost of such immunisation in the London Schools, as estimated by Dr. J. Graham Forbes,[1] would be between £85,000 and £100,000. During 1921 the cost of diphtheria to the rate-payers was £500,000, and the cost of each case of diphtheria £30, a sum which would cover the cost of protecting 200 children. There would thus be not only an immense saving of life but also a saving of about £400,000 a year.

Mode of Action of Toxin-antitoxin.—When diphtheria toxin is artificially introduced into the system it stimulates the formation of antitoxin. If, however, a dose of toxin large enough to produce a sufficient dose of antitoxin to immunise the person were to be injected, it might be accompanied by considerable risk. But experiments with guinea-pigs have shown that when the toxin is neutralised with antitoxin it loses its poisonous properties, although it still retains its capacity for stimulating the formation of antitoxin.

The following figures show the value of such immunisation in the case of the staff attending diphtheria cases in hospitals in Birmingham (Harries):—

TABLE CLXXXIV.

Year	Average number of diphtheria cases admitted per annum	Average number of female staff at work	Average number per annum of cases of diphtheria among staff	Remarks
1916-22	941	123	18 (= 14 6 per cent.)	No immunisation
1924	1,734	153	6 (= 3·9 ,,)	Active immunisation of nursing staff
1925	2,005	125	1 (= 0·[illegible] ,,)	Do. do.

Similar statistics have been published for Edinburgh and Aberdeen and other parts of the world. On the other hand, in hospitals where the staff have not been immunised no such drop has occurred. The following figures show the reduction of the diphtheria incidence in New York and Chicago since the introduction of the Schick test and active immunisation:—

[1] "The Prevention of Diphtheria," Medical Research Council Special Report, No. 115 (1927).

TABLE CLXXXV.

Locality	Year	Number of cases notified	Number of deaths	Number of persons Schick-tested	Number of persons immunised
(*a*) New York City	1919	14,014	1,239	12,000	5,150
	1920	14,166	1,045	20,000	8,000
	1921	15,110	891	140,560	42,000
	1922	10,427	873	146,840	48,496
	1923	8,050	553	119,493	42,818
	1924	9,687	714	130,493	58,936
	1925	9,051	663	83,854	38,191
(*b*) Chicago ..	1922	7,367	564	5,413	8,396
	1923	5,836	366	18,470	28,378
	1924	3,672	216	23,296	38,276
	1925	2,926	239	21,579	45,282

The Older Methods of Combating Diphtheria.—The two methods most commonly employed are :—

(1) Isolation of all contacts, the swabs from whose throats showed the presence of the Klebs-Löffler bacillus (K.L.B.); (2) the injection of a prophylactic dose of antitoxin (500 units for a child and 1,000 units for an adult) to all contacts; this produces temporary immunity in twenty-four hours.

Neither of these methods is ideal, for the following reasons :—

(*a*) Those isolated under the first method will include several groups of contacts, viz. :—

(i) Those who are susceptible (i.e., Schick-positive) consisting of: (α) cases whose swabs show avirulent bacilli; (β) cases whose swabs show virulent bacilli.

(ii) Those who are insusceptible (i.e., Schick-negative) consisting of: (α) cases whose swabs show avirulent bacilli; (β) cases whose swabs show virulent bacilli.

(The distinction between virulent and avirulent bacilli is made by testing them on guinea-pigs. Only the virulent ones will produce diphtheria when injected into guinea-pigs.)

Class (ii) (α), being themselves insusceptible and harbouring avirulent bacilli only, are in no danger to themselves or to others and therefore require no isolation.

Class (i) (α) is really not a true "carrier," but being a susceptible person his association with those

carrying the virulent bacilli [whether Schick-positive, Class (i) (β), or negative, Class (ii) (β)], involves considerable danger of his catching the disease, especially from (i) (β), who is obviously incubating diphtheria.

(*b*) The injection of antitoxin to all contacts, under the second method, is objectionable on two grounds, viz.:—

(i) The immunity conferred is exceedingly transitory and disappears in a very few weeks.

(ii) The injection renders the patient's serum sensitive, and should it be necessary to give him another injection, if ever he gets diphtheria, the risk of anaphylaxis will arise, although the risk is very slight indeed.

These objections are eliminated by the method of immunisation, since not only are the susceptible separated from the insusceptible, but the susceptible ones are given an immunity which lasts a number of years. There is, however, one drawback to the modern method of immunisation, viz., its slowness, since it takes a few weeks to confer the necessary immunity, and is, therefore, not applicable in cases of emergency.

Emergency Immunisation.—The following scheme for rapidly controlling an epidemic in an institution whose inmates had not previously been Schicked and immunised has been devised by Okell, Eagleton and O'Brien[1]:—

(1) Isolate the patient and carefully disinfect all articles used by him, such as utensils, bedding, clothing, &c.

(2) Swab throats and noses of all persons.

(3) Schick test all persons in the institution.

(4) After twenty-four hours **isolate** all Schick-negative persons whose swabs are positive until the virulence of the bacilli has been ascertained, and release all the "avirulent" carriers, since they are not dangerous either to themselves or others. The Schick-negatives, however, who are "virulent" carriers, must be rigidly isolated, for although they are themselves immune they are a danger to others.

(5) Begin to immunise at once all Schick-positives. The problem of the virulent **diphtheria carriers** is a very difficult one, since it is obviously impossible to keep them isolated indefinitely. Tonsillectomy may abolish the condition, if the bacilli reside in

[1] *Lancet*, i, 1924, p. 800.

the tonsils, but not in others in whom the bacilli may reside in the nasal sinuses or other situations. Under universal immunisation the difficulty would never arise, since such carriers would obviously become innocuous to an immunised population. It has been estimated by Savage that about 2 to 3 per cent. of the general population are diphtheria carriers, whilst of carriers of virulent bacilli, the class that really matters, there are no more than about 0·2 per cent.

Scarlet Fever.

In scarlet fever (first described in 1675 by Sydenham, who gave the disease its name) the infection is not a very diffusible one, and hence isolation, quarantine and disinfection have failed to limit the spread of the disease. Sir John Robertson has come to the definite conclusion that isolation in special hospitals is of very little value, and if we could have perfect bed isolation under intelligent supervision scarlet fever cases could safely be treated at a general hospital or at home. Medical officers of large residential schools have found it very difficult to check an outbreak of scarlet fever by isolation and quarantine, since many such outbreaks are due to cases that are so mild as to be unrecognisable.

The following figures give the numbers of notifications for various ages in Detroit, in 1920:—

TABLE CLXXXVI.

Age-period	0-	1-	2-	3-	4-	5-	6-	7-	8-	9-
Notifications per cent. ..	0·6	2·4	4·9	7·8	10·3	10·2	11·1	9·3	7·3	5·3

In other words, the highest incidence is among children between 3 and 8 years of age, and about 70 per cent. of all notifications are before the age of 10.

TABLE CLXXXVII.

	1861-70	1924
All ages	857	27
0-	2,026	37
1-	5,172	129
2-	5,907	156
3-	5,687	169
4-	4,667	143
0-	4,644	128
5-	1,394	45
15-	124	9
25-	61	4
35-	28	3
45-55	14	2

The decline in mortality from scarlet fever is shown by the preceding table CLXXXVII.

The following table shows the stages of the reduction :—

TABLE CLXXXVIII.

	All ages	0-5 years
1861-70	1,000 (Standard)	1,000 (Standard)
1871-80	729	755
1881-90	350	359
1891-1900	177	182
1901-10	127	123
1924	32	28

We see, therefore, that the mortality in 1924 was only 3 per cent. of that of 1861-70.

The Dick Test.—In January, 1924, Dr. and Mrs. Dick introduced a skin test (similar to the Schick test) for the susceptibility to scarlet fever, and preventive immunisation against the disease by injecting small amounts of scarlet fever toxin in susceptible cases. The following results have been found by Abraham Zingher,[1] of New York, after applying the Dick test :—

Of infants under 6 months	45 per cent. are susceptible
Of children, 1-2 years	71 ,, ,, ,,
Of children, 15-20 years ..	Only 17 ,, ,, ,,

Bokay found the Dick test to be negative in sixty-two out of sixty-six new-born infants.

Dr. Harries, of Birmingham, gives the following figures :—

TABLE CLXXXIX.

Age	No previous history of scarlet fever
0-5 years	70 per cent. are susceptible
5-10 ,, ...	64 ,, ,, ,,
10-15 ,, ...	56 ,, ,, ,,
15-20 ,, ...	18 ,, ,, ,,
Over 20 years	11 ,, ,, ,,
All ages	47 5 ,, ,, ,,

New-born babies are Dick-negative if the mothers are Dick-negative.

Immunisation.—Several thousands of children in hospitals and infant welfare centres in New York, who have been found to

[1] *Journ. Am. Med. Assoc.*, 1924.

give a positive reaction to the Dick test, have been inoculated with scarlatinal hæmolytic streptococcus toxin, three inoculations having been given intramuscularly at weekly intervals. Of 274 such inoculated children, 167 (i.e., 61 per cent.) gave a negative Dick test four to five weeks after inoculation, and no child that has given a negative Dick reaction on admission to the hospital has contracted scarlet fever, although admitted into scarlet fever wards. The Dick test and the method of immunisation are obviously too recent to enable one to draw definite conclusions, and for the present one must still rely upon isolation and disinfection as the methods of preventing the disease.

As a substitute for hospital isolation, or in cases where scarlet fever patients are treated at home, one may adopt the Milne method, which consists in anointing the child with eucalyptus oil (see fig. 145, on p. 568).

The accuracy of the Dick test has been confirmed in Moscow, where it has been found that the notification curve of scarlet fever per 10,000 children living at each age-period ran a parallel course with the percentage curve of positive Dick reactions at the corresponding ages.[1]

The Schick and Dick tests have shown that a larger percentage of school children in the richer schools are susceptible to diphtheria and scarlet fever than in the poorer schools ; probably the former children live more isolated lives than the latter.

Relation between Overcrowding and the Incidence of Scarlet Fever.—Brownlee[2] has shown that the incidence of scarlet fever in houses accommodating not more than one person per room was about three times as high as in houses where the density was 2·5 to 2·75 persons per room, as is seen from the following table for Glasgow (1898-1902) :—

TABLE CXC.

District	Population	Number of children under 10 years	Number of people per room	Scarlet fever cases	Incidence per 10,000 children
I	34,868	4,451	0·5-1·0	864	388·2
II	83,255	13,605	1·0-1·5	2,148	318·6
III	201,0 8	43,173	1·5-2·0	5,439	251·8
IV	87,885	20,834	2·0-2·25	2,186	209·6
V	237,161	56,898	2·25-2·50	5,610	197·0
VI	117,445	30,185	2·50-2·75	2,091	138·0

1 *Journ. Amer. Med. Assoc.*, 1925, i, 1476.

2 Medical Research Council Special Reports Series, No. 18.

These figures, therefore, afford strong evidence in favour of the view that active immunisation is obtained by frequent exposure to mild cases.

The Schultz-Charlton Reaction for the Diagnosis of Scarlet Fever.—If serum from a convalescent scarlet fever patient or from an immunised horse be injected intradermally into the skin of a patient suffering from a rash suspected to be scarlet fever, there will, in a large number of cases, be a blanching of the rash over the area of injection in the course of six to eight hours if, and not unless, the case is one of scarlet fever. The test is most reliable on the first day, when it gives a correct diagnosis in some 80 per cent. of the cases.

Measles.

The infection in measles is very diffusible, but hospital isolation is of little value in checking its spread, mainly because the period of greatest infectivity occurs before the disease is recognised. Disinfection in the ordinary way (by means of sulphur fumes) is also practically worthless, as the infective agent dies very rapidly on exposure to light and air. Sir John Robertson is satisfied that there is no fear of a child *who has previously had measles* spreading the disease in a school when coming from a house in which there is a case of measles.

In infants' schools the rate of spread of measles depends largely upon the ventilation. In badly-ventilated schools one case will infect nearly all the children, whilst in open-air schools the disease hardly spreads at all.

Measles is a formidable disease, and during the past ten years it has killed on an average 3,500 children per year in England and Wales—more than any other infectious disease of childhood. But although the death-rate from this disease has been recently declining the incidence-rate has not declined to the same extent.

TABLE CXCI.—MEASLES MORTALITY BETWEEN 1861 AND 1920 (IN ENGLAND AND WALES) ACCORDING TO AGE.

	0-1 per 1,000 births	1-2	2-3	3-4	4-5	5-10
		per million living				
1861-70	2·3	6,243	3,236	1,730	968	243
1871-80	2·3	5,411	2,465	1,389	778	208
1881-90	2·9	6,673	2,916	1,684	1,031	271
1891-1900	3·1	7,097	2,861	1,593	960	221
1901-10	2 6	5,830	2,224	1,227	753	174
1910-20	2·4	5,348	2,369	1,241	775	220

Immunisation.—In 1916 Dr. Ch. Nicoll, of the Pasteur Institute in Paris, discovered the fact that the blood-serum of patients convalescent from measles will definitely cut short the disease if injected into individuals during the early incubation stage. Professor Debré and Dr. Joanon, of Paris, have taken up this work, and have shown that a temporary immunity from the disease can be obtained in this way. By this means measles is said to have been entirely eliminated from the hospital crêches in which formerly numbers of children were attacked, with a heavy resulting mortality. Professor Debré has further shown that the serum of any adult who has previously had measles, even in early childhood, is capable of conferring immunity against the disease in a child. If substantiated, this is a most important discovery, because the child's parents' blood may be used, thus obviating any difficulty arising from trying to find a donor belonging to the same blood-group as the patient. Three cubic centimetres of the serum suitably sterilised are injected subcutaneously into a child of 3 years of age, and, provided the injection is done under aseptic precautions, is not attended by any risk. The protection, however, that it affords is only very temporary, not lasting more than about a fortnight, but this is long enough to avert the danger of immediate infection which is so valuable in the case of young children exposed to the disease. There is evidence to the effect that the earlier in the incubation period the serum is administered the more certain the immunity, and that when injected late in the incubation period, the likelihood of evading an attack is very small, but the attack is most probably going to be very mild. It has therefore been advised to inject the serum, not immediately after exposure but after a lapse of about a week from the time of exposure, in order to give the patient a mild attack of the disease which will confer on him permanent immunity. Indeed Hermann, of New York, has recently tried to produce an active immunity in infants by producing mild measles infection in them before their passive congenital immunity has disappeared. The material he uses is the nasal discharge from a case of measles, mixed with saline and centrifuged, which he applies to the nasal mucous membrane of the baby.

It must be remarked, however, that the whole subject of immunisation against measles is still in its experimental stage.

J. H. Townsend[1] finds that 20 c.c. of the blood of an

[1] *Boston Med. and Surg. Journ.*, May 13, 1926, p. 869.

adult who had had measles twenty years previously was utterly useless in preventing or modifying the disease. But 9 c.c. of the blood (about 5 c.c. of serum) from a convalescent, whilst unable to prevent infection, modified the disease if it was administered before the end of the first week of the incubation period.

Immunity at Birth.—New-born babies of mothers that had had measles are themselves immune, and this immunity persists for about four to five months. As about 90 per cent of the community are known to have had measles, at least 90 per cent. of the new-born babies have a temporary congenital immunity.

Whooping-cough.

Whooping-cough, most probably caused by the Bordet-Gengon bacillus, is said to be preventable in the same way as measles, i.e., by injecting the serum of a patient who has had the disease for five or six weeks.

Acute Anterior Poliomyelitis (Infantile Palsy).

Infantile palsy occasionally occurs in epidemic form, but the exact mode of its spread is not known with certainty. It seems, however, to be less infective than either diphtheria or scarlet fever. The work of Flexner and Clark, in 1911, has shown that large doses of hexamine are effective in preventing the disease in experimental monkeys. When the drug is given in large doses by the mouth its presence can be detected in the cerebro-spinal fluid soon afterwards, and thus acts as a local antiseptic to the virus of the disease. Arthur F. Hurst[1] suggests that this observation may be utilised in the prevention of the spread of the disease during an outbreak. Hexamine (urotropine) in doses of 100 gr. (7 grm.) is to be given three times a day. As, however, the drug is decomposed in acid media with the liberation of formalin, which may therefore in its excretion by the bladder set up cystitis and hæmaturia, it is necessary to render the urine very alkaline (in which the hexamine does not break up) by giving at the same time sufficient alkali, viz., 60 gr. each (0·4 grm.) of bicarbonate of soda and potassium citrate in an ounce of water. The alkali will not affect the action of the urotropine on the cerebro-spinal fluid, which is always alkaline.

[1] "The Prophylaxis of Poliomyelitis," *Brit. Med. Journ.*, December 4, 1926.

Chicken-pox or Varicella.

The following figures show the great incidence of the disease in early childhood. In Basle, in 1875-1906, out of 6,014 reported cases there were :—

691	cases in infants under 1 year old	= 11·3 per cent.
5,080	,, ,, children over 1 year old	= 84·4 ,,
21	,, ,, people between 20 and 40 years of age.	

It is possible to immunise children against chicken-pox by inoculation with the matter obtained from chicken-pox vesicles, or by injecting serum from the blood of patients convalescent from the disease, but, owing to the mildness of the disease, immunisation has not been extensively used.

Mumps.

In view of the usually very mild nature of the disease and our ignorance of its cause, immunisation against it has not been practised to any extent, although injection of serum from patients convalescing from the disease has, in Hess' hands, given encouraging results. For the present isolation is the only method of prevention available.

The School in Relation to Infectious Diseases.

We have seen that the infectious diseases are commonest at school age, and the question therefore arises, "Does the school play an important part in spreading infection?" The following facts bear upon this question :—

(1) Sir Shirley Murphy noticed that "a fresh factor of diphtheria at the ages 3 to 10 years became operative in the decennium 1871-80." As the Elementary Education Act first became operative in 1871, it is likely that the school attendance may have caused a spread of the disease.

(2) Murphy further noticed, in 1894, that depressions occurred in the London incidence curves of scarlet fever and diphtheria about a week after the closure of schools for the summer holidays, which is the approximate duration of the incubation period. There was no similar depression in the typhoid curve.

(3) The holiday depression occurred particularly in the curve for the age-period 3 to 13 years, i.e., the school age. As there is no reason to suppose that children of this age migrated from London during the school closure season much more than people of other ages, it points to the school being a factor in the spread of these diseases.

On the other hand, the fact that the greatest prevalence of infectious disease occurs during the age-period 1 to 5, i.e., the pre-school rather than the school age, is a point against the theory of school infection. Indeed, we can say quite definitely that infectious diseases are considerably less common during school age than during the pre-school period.

Nevertheless, there can be no doubt that owing to the close association of children in the class-room, the school must play as great a part in the dissemination of infectious diseases amongst the scholars as many other indoor places where children congregate, such as cinemas, &c.

TABLE CXCII.

Disease	Exclusion periods	
	Affected children	Children in infected houses
Scarlet fever and diphtheria	14 days after discharge from hospital or disinfection of house, if child was treated at home	14 days from certification if removed to hospital, or 7 days from disinfection of house, if child was treated at home
Measles	3 weeks from date of appearance of rash	If child has had measles may attend at once, if not, 14 days after incidence of last case
German measles	1 week from date of rash	As for measles
Mumps	7 days from subsidence of swelling	May attend school but should be watched
Whooping-cough	As long as cough continues and not less than 6 weeks from onset.	If child has had the disease may attend at once, if not, 21 days from onset of last case
Chicken-pox..	Until all traces of scales have disappeared	As for whooping-cough
Small-pox	After discharge from hospital	16 days

Should Schools be Closed during an Epidemic?

The point in favour of such a procedure is obviously the fact that the uninfected children would not be brought into contact with those incubating the disease. On the other hand, the fact that the teacher can notify any new case as it occurs is a reason why schools should not be closed. Indeed, experience has shown that school closure has not been effectual in stopping an epidemic,

at any rate in urban districts where the children live close together and have every opportunity for mixing together and spreading the infection without supervision. The Board of Education are of opinion that school closure is "a measure that seldom ought to be enforced. . . ." The best method of preventing the spread of infection during an epidemic is the careful exclusion of individual children. The L.C.C. have issued regulations regarding the duration of exclusion of children from their schools in cases of infectious disease which are summarised in Table CXCII.

The American Public Health Association in 1917 requested the Federal Bureau of Education to report on the matter, and the result of the investigation and deliberation is that: "Closure of schools as a means of controlling epidemics of measles, whooping-cough, scarlet fever, diphtheria, small-pox and poliomyelitis is unnecessary, unscientific and unjustifiable. That disinfection by fumigation is unnecessary and ineffective. . . . Disinfection by air and sun and cleansing with hot water, soap, and scrubbing, is to be recommended. . . ."

During the influenza epidemic of 1918, New York City, in contradistinction to other places in the United States, abstained from closing its schools, but instituted the following procedure, viz.:—

(1) Upon arrival at school all children went directly to their class-rooms without coming into contact with children of other class-rooms.
(2) Class intervals were held at different times for the different classes, and group games were allowed between children of the same class-room.
(3) On leaving school the children went straight home without mixing together.
(4) Each morning the children were inspected by their teachers, who had been trained to detect early symptoms and signs of the disease, and any suspicious case was immediately sent home, where it was visited regularly by the school doctor or nurse.

As a result of this intensive supervision, the absence-rate of children from school during the entire influenza period was not greater than during the corresponding months of previous years. Indeed, the incidence and mortality-rate from influenza of children 5 to 15 years old was not higher during the epidemic than during the same months of other years.

Small-pox.

In view of the considerable controversy that has been raging on the question of the efficacy of vaccination against small-pox, it seems to the author advisable to give a brief history of the prevalence of this disease before and after the introduction of vaccination by Edward Jenner, in 1798, and to discuss the various objections that have been raised against this simple operation.

History.—It is probable that the disease prevailed in India and China over two thousand years ago, but the earliest medical description of the disease was given by the Arabian physician Rhazes, in the early part of the tenth century, who mentions that Galen was familiar with it. The accounts found in the annals of the various monasteries reveal the presence of this disease in Europe in the Middle Ages. It was brought from the East to the West by the Crusaders in the eleventh and twelfth centuries. In England the disease was well known in the Elizabethan period, and the London Bills of Mortality, first compiled by the order of Thomas Cromwell in 1558, but systematically begun to be kept in 1593, show that small-pox was prevalent in London at that time. Sir John Simon, who studied these records, found that the small-pox mortality in London between 1660 and 1679 was 4,170 per million. The diaries of John Evelyn and Samuel Pepys, as well as the letters of Horace Walpole, show the great prevalence of the disease in the later part of the sixteenth century. It struck down prince and peasant, rich and poor alike. Evelyn records the death of his own two daughters, as well as of the prospective husband of one of them; of the Archbishop of York (April 15, 1686), and of Queen Mary, wife of William III (December, 1694). The king's parents, uncle and two cousins also died of the disease, and the king himself escaped with his life but with a permanently damaged constitution. Under the date January 13, 1695, Evelyn has the following entry: "The deaths in London by small-pox increased to 500 more than in the preceding week." With a population of 400,000—the population of London at that time—such a mortality means a death-rate for the week of 1,250 per million. It is interesting to contrast this figure with the mortality during the London epidemic of 1901-02, which was at the rate of twenty a week out of a population of 5,000,000, i.e., 4 per million. In other words, the small-pox mortality in 1695 was about three hundred times as high as during the recent epidemic. Between 1906 and 1920 the small-pox mortality for the whole of England was less than 1 per million per annum. During the eighteenth century the disease, according to the famous mathematician, Daniel Bernouilli carried off about one-thirteenth of each generation, including many European monarchs, among whom may be mentioned Joseph I of Austria (1711), Louis XV of France (1774), and Peter II of Russia (1730).

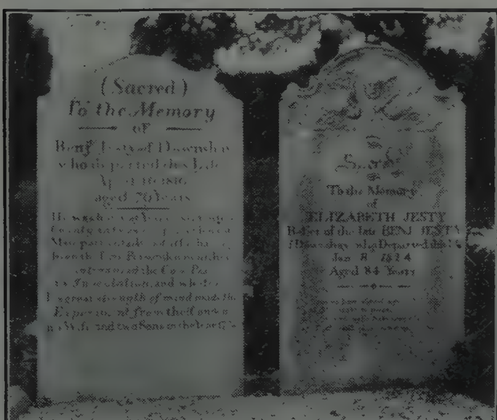
Dr. Robert Watt, writing in 1813 on the relative mortality from the principal diseases of children between 1783 and 1812, states that more than half of the human species died before they were 10 years old, and of this half more than a third died of small-pox.

The disease was equally prevalent in other parts of the world. According to Prescott ("History of Mexico"), the ravages of small-pox in 1520 were

terrible. It swept over the land "like fire over prairies. . . . So great was the number of those that died of the disease that there was no possibility of burying them and the bodies were thrown into the canals . . . till the air was poisoned with the stench of putrid bodies."

In 1707 an epidemic occurred in Iceland which is said to have killed 18,000 out of 50,000 inhabitants, a death-rate from that disease alone of 36 per cent. These figures, owing to defective registration at the time, may not be perfectly accurate, but they are probably not far from the truth. The number of such examples can be greatly multiplied, but it will suffice to quote Lord Macaulay, who wrote of small-pox that it was "the most terrible of all ministers of death, always present, filling the churchyards with corpses, tormenting with constant fears all whom it had not yet stricken, leaving on those whose lives it spared the hideous traces of its powers, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the betrothed maiden objects of horror to the lover." One may also quote Ben Jonson's couplet:—

> "Envious and foule disease can there not be
> One Beautie in an Age and free from thee?"



[By kind permission of the British Medical Journal.

Fig. 146.—Benjamin Jesty's grave.

Vaccination.—It was noticed by dairymen and others that persons who had suffered from cow-pox, contracted by milking cows affected with the disease, were immune against small-pox, and in 1774 a Dorsetshire farmer, Benjamin Jesty by name, was, as recorded on his tombstone (fig. 146), the first person known to have successfully inoculated his wife and two sons with cow-pox matter, and Peter Plett, a tutor of Holstein, successfully vaccinated three of his employer's children in 1791. The credit, however, of intro-

ducing vaccination as a scientifically established preventive remedy against the disease is entirely due to Edward Jenner (1749-1823). Jenner was a student and protégé of the famous John Hunter, and he wrote to his master regarding his observation that Gloucestershire dairymaids who contracted cow-pox were immune against small-pox, and that he was thinking about the matter. Hunter replied: "Do not think, try; be patient, be accurate." Jenner made a number of observations and experiments from 1788 until 1796, when he inoculated a boy (Phipps) with cow-pox material, using pus from a dairymaid infected with cow-pox. Subsequent application of the "variolous test," i.e., inoculation with small-pox material, a few weeks and a few months later, failed to infect the boy with small-pox. In 1796 Jenner inoculated a child with cow-pox material; from this child he inoculated another child, and so on from child to child, and the variolous test proved negative in three of the children to whom the test was applied. In 1798 Jenner published his epoch-making book, "An Inquiry into the Causes and Effects of Variolæ Vaccinæ," and thus furnished medicine with what is one of her greatest victories. He was given by Parliament a grant of £20,000 to carry on his experiments.

Vaccination was put to the crucial test in Boston, in August, 1802, when nineteen boys were vaccinated. The variolous test applied to them was negative in every case; all the unvaccinated boys inoculated (as controls) with the same small-pox virus took the disease. "All over the civilised world," as McVail remarks, "thousands of persons who had not undergone a certain preliminary operation were inoculated with the virus of small-pox, and with a very small percentage of failures all received the disease; and simultaneously all over the civilised world thousands of persons who had undergone the preliminary operation in question were also inoculated with the virus of small-pox, and with a very small percentage of exceptions all resisted the disease." We thus see that the value of vaccination has been firmly established on a scientific basis as the result of thousands of exact experiments on human beings.

There is no doubt that but for the genius of Edward Jenner, small-pox, instead of being practically extinct, would almost certainly still have been a universal scourge, as it still is in certain parts of the world.

The Influence of Vaccination on the Incidence of and Mortality from Small-pox during the Nineteenth Century.—Reliable records

[*From Professor Hope's "Public Health" (Livingstone) by kind permission.*

FIG. 147.—Small-pox rash in a woman who had never been vaccinated.

[*By kind permission of Professor Hope and Messrs. Livingstone.*

FIG. 148.—Modified small-pox in a vaccinated mother with vaccinated baby in her arms free from small-pox.

of small-pox deaths during the first quarter of the nineteenth century are found in the London Bills of Mortality, in Sweden and Copenhagen, where accurate statistics have been kept since 1774 and 1750 respectively, and in other countries. In Sweden and Copenhagen there was a fall in small-pox mortality from 1801 onwards. Indeed, the Royal Commission on Vaccination, in their Final Report, say that: "The records of Western Europe and the United States show that in all places whence returns were obtained, the introduction of vaccination was followed by a decline of small-pox." It may further be added that in countries where vaccination did not become general there was no such decline in small-pox.

Statistics of Vaccination and Small-pox.

The following statistics show the mortality from small-pox per million population, before and soon after vaccination :—

TABLE CXCIII.

	Before vaccination	After vaccination
Sweden	2,050 per million	158 per million
Prussia	5,593 ,,	912 ,,
Austria	3,905 ,,	841 ,,
Berlin	3,422 ,,	176 ,,

In the case of Sweden particularly, the statistics are very accurate, since exact records have, as we have already said, been kept there since the year 1774. The following are more detailed figures for Sweden :—

1773-1800 .. .. .. (no vaccination)	Average small-pox mortality was	2,008 per million
1801-1815.. .. .. (permissive vaccination)	,, ,, ,, ,,	631 ,,
1816-1885 .. .. .. (compulsory vaccination)	,, ,, ,, ,,	173 ,,
1893-1896 .. .. .. (improved administration of vaccination law)	,, ,, ,, ,,	2 ,,

The Copenhagen figures are as follows :—

TABLE CXCIV.

Years	Mortality per million
1750-1800 (no vaccination)	3,567
1800 onwards (permissive vaccination)	660
1810-70 (compulsory vaccination)	130
1893-97 ,, ,, ...	0·5

In Germany and Switzerland there are places where at certain given periods vaccination was compulsory in some and not compulsory in others. The following statistics compare the small-pox mortality in those areas :—

TABLE CXCV.

	Germany (1875-83)		Switzerland (1880-92)	
	Dresden (compulsory vaccination)	Prague (no compulsory vaccination)	Cantons with compulsory vaccination	Cantons without compulsory vaccination
Average mortality ..	1·6	143	9·4	35

These various statistical tables will serve as examples of the numerous similar statistics in different parts of the world. They all illustrate the same facts, viz. : (1) Epidemics of small-pox are very much rarer and milder now than before the vaccination period ; (2) in places where vaccination and revaccination at school age are compulsory, small-pox is rare, and its fatality-rate is low. It is otherwise in places where vaccination is not enforced, unless those places happen to be either isolated communities like New Zealand, or surrounded by well-vaccinated communities, e.g., Leicester.

TABLE CXCVI.

Before vaccination	Vaccination encouraged but not compulsory		Vaccination nominally compulsory but not stringently enforced		Vaccination compulsory and enforced	Revaccination of school children	Thrice vaccinated
Sweden, 1792-1801	Prussia 1860-69	Austria 1881-90	Sweden 1859-68	England 1857-66	England 1889-98	Germany 1890-99	Prussian army 1875-99
880	190	814	388	206	0·8	1·2	Two deaths in twenty-five years. One death refers to a reservist twice unsuccessfully vaccinated
944	302	948	183	335	0·6	1·0	
1,758	211	592	49	197	1·7	2·1	
2,956	338	508	37	140	14·7	3·1	
1,963	463	601	76	66	49·3	1·7	
751	438	382	182	81	27·3	0·5	
585	620	447	325	293	7·3	0·2	
1,609	432	645	293	373	17·6	0·1	
5,126	183	557	253	309	0·8	0·3	
2,566	194	249	342	144	8·1	0·5	
Average in 10 years, 1,914	337	574	213	214	13·0	1·0	

The preceding table shows the differences in small-pox mortality rates (per million population) under different conditions of vaccination in different countries. The statistics are gross and give the total mortalities, without discriminating the vaccinated from the unvaccinated individuals.

It is to be remembered that the first law for compulsory vaccination of infants in Prussia came into force in 1874, and it enacted that children should be vaccinated before they were 2 years old. It also made revaccination compulsory at school age, and again when being recruited for the army.

In Italy the vaccination laws date from 1888 and 1891, and while the small-pox mortality was 355 per million in 1881-90, it dropped to 53 in 1904-08, only fifteen years after the introduction of compulsory vaccination.

The statistics so far given merely show that there has been a close association between vaccination in different parts of the world and the incidence of mortality from small-pox in those parts, and although they strongly suggest a close causal relationship between the two, do not necessarily prove it, since the decline in small-póx might conceivably have been due to some other cause which might have become operative at the beginning of the nineteenth century (see Chapter III, p. 80 *et seq*., and also below, p. 608 *et seq*). In order to investigate the causal relationship between vaccination and the decline of small-pox, we must study on a large scale the behaviour of *vaccinated individuals* during small-pox epidemics, as compared with *unvaccinated individuals* during the same epidemic in the same place and living under the same conditions with regard to social and economic environment. Such statistics exist in large numbers, and they invariably prove that vaccinated people are less susceptible to small-pox than unvaccinated people, and that when they do get the disease the fatality-rate amongst them is less than among the unvaccinated.

TABLE CXCVII.—FRENCH STATISTICS SHOWING THE INCIDENCE OF, AND MORTALITY FROM, SMALL-POX AMONGST VACCINATED AND UNVACCINATED INDIVIDUALS.

	Vaccinated		Unvaccinated	
	Incidence	Mortality	Incidence	Mortality
Whole of France in 1802 ..	1 in 317	1 in 7,166	1 in 12	1 in 40
Marseilles in 1825	1 ,, 15	1 ,, 1,500	1 ,, 2	1 ,, 8

In Marseilles, in the same year, out of 2,000 who had previously had small-pox, there were twenty cases with four deaths, giving an incidence of 1 in 100 and mortality of 1 in 500.

The preceding statistics from France and different parts of England illustrate this fact.

The following are English statistics relating to an epidemic in 1887-88 :—

TABLE CXCVIII.

	Attack-rate under 10		Attack-rate over 10	
	Vaccinated	Unvaccinated	Vaccinated	Unvaccinated
Sheffield	7.9	67·6 (8·5 : 1)	28·3	53·6 (2·0 : 1)
Warrington	4·4	54·5 (12·7 : 1)	29·9	57·6 (2·0 : 1)
Dewsbury	10·2	50·8 (5·0 : 1)	27·7	53·4 (2·0 : 1)
Leicester	2·5	35·3 (14·0 : 1)	22·2	47·6 (2·0 : 1)
Gloucester	8·8	46·3 (5·0 : 1)	32·2	50·0 (1·5 : 1)

The figures in brackets represent the incidence ratio between unvaccinated and vaccinated.

Dr. Coupland's statistics of small-pox in vaccinated and unvaccinated in the *same invaded houses*, where all social, hereditary and environmental influences are as nearly identical as they ever can be, probably afford the best proof regarding the immunising effects of vaccination. Here are the statistics for children under 10 years of age :—

TABLE CXCIX.

	Incidence per cent.	
	Vaccinated	Unvaccinated
Leicester	2·5	35·3
Gloucester	8·8	46·3
Dewsbury	10·2	50·8

To say, as the opponents do, that the unvaccinated are so because they were too ill to be vaccinated and would therefore naturally succumb more easily to small-pox, is not true, since returns show that in Whitechapel, for instance, out of 2,092 available births during a certain year, 1,663 were successfully vaccinated, and only 4 out of the remaining 429, i.e., less than 1 per cent. of the unvaccinated, were postponed by medical certificate.

The following are some more recent statistics. In West Ham, in 1921, there were 46 cases of small-pox in children under 5; of these, 45 had never been vaccinated. In Glasgow, in

1920-21, there were 112 cases of small-pox in unvaccinated children under 15, with 38 deaths (fatality-rate, 34 per cent.), and only 30 cases among vaccinated children under 15 (most of them vaccinated in infancy and none of them revaccinated) with not one death.

TABLE CC.—TYPE OF DISEASE IN VACCINATED AND UNVACCINATED.

	Mild		Severe	
	Vaccinated	Unvaccinated	Vaccinated	Unvaccinated
Sheffield ..	82·8 per cent.	18·5 per cent.	17·2 per cent.	81·5 per cent.
Dewsbury ..	82·0 ,,	23·1 ,,	18·0 ,,	76·9 ,,
Leicester ..	81·4 ,,	27·2 ,,	18·6 ,,	72·8 ,,
Warrington..	78·2 ,,	29·4 ,,	21·8 ,,	70·6 ,,

Further, the revaccinated postmen, in spite of their constant contact with small-pox houses, never get small-pox during an epidemic, whilst the members of their families who live under the same domestic environments and do not come into such contact do get the disease.

Moreover, statistics show that the more recent the vaccination the more immune is the individual from death or even from attack by small-pox. Thus, in the case of Sheffield, out of 33,393 vaccinated children under 5 years of age, 121 took small-pox and one died, giving an incidence-rate of 0·33 per cent., a mortality-rate of 0·003 per cent., and a fatality-rate of 0·8 per cent. On the other hand, of the 1,981 unvaccinated children under 5 years, 128 took the disease and 66 died, giving an incidence-rate of 6·5 per cent., a mortality-rate of 3·3 per cent., and a fatality-rate of 51·6 per cent. In other words, the incidence-, mortality-, and fatality-rates amongst the unvaccinated children were 20, 1,100, and 65 times as high respectively as amongst the vaccinated children *in the same epidemic.*

We see, therefore, that in the case of the Sheffield epidemic the incidence and mortality from small-pox varied inversely with the recency of vaccination. The following table shows the incidence-rates in vaccinated and unvaccinated respectively :—

TABLE CCI.

Incidence ratio under 5		Incidence ratio under 10		Incidence ratio over 10	
Vaccinated	Unvaccinated	Vaccinated	Unvaccinated	Vaccinated	Unvaccinated
1	20	1	8·5	1	2

From this we learn that the immunity afforded by vaccination is not permanent, but lasts at any rate for at least five years. It rarely disappears completely, although it sometimes does, as does also the immunity conferred by an actual attack of the disease itself. In the Leicester epidemic of 1903, for instance, not a single case occurred amongst vaccinated children under 10, and not one death occurred amongst vaccinated persons under 40 years of age. Of the unvaccinated, 81 cases occurred in children under 10 years, with a mortality of 10 per cent. Hence we see that after a lapse of a certain number of years **revaccination** becomes necessary; this is especially the case during an epidemic.

The value of revaccination is shown by the immunity of the revaccinated medical, nursing and domestic staffs of small-pox hospitals. There are many figures which prove this, and the experience of Mile End Infirmary in the 1902 epidemic may be cited as an example. Out of forty-two members of the nursing staff, thirty-five had been revaccinated. Of these not one contracted small-pox. Of the remaining seven three were vaccinated during an advanced stage of small-pox incubation and sickened with the disease two, three and six days respectively after vaccination, and the other four all got small-pox.

The value of revaccination, as illustrated by the German army during the Franco-Prussian War, has already been shown in Table CXCVI, on p. 597.

This is especially striking if one compares the incidence and mortality from small-pox in the well-vaccinated German army with: (*a*) the unprotected French army during the same war; (*b*) the well-vaccinated French army during the Great World War.

During the Franco-Prussian War there were in the German army 5,000 cases of small-pox (6·25 per 1,000), with 300 deaths (fatality-rate 6 per cent.), as compared with 125,000 cases in the French army, with 23,470 deaths (fatality-rate 19 per cent.). If small-pox had occurred in the French army during the Great War with the same frequency as in 1871-72, there would have been some 1,200,000 cases with 225,000 deaths. As a matter of fact, owing to strict enforcement of vaccination and revaccination, there were no more than 66 cases, with 5 deaths (fatality-rate 7·5). In the German army, during the same war, there were 434 cases (0·0111 per mille), with a fatality-rate of 4·6 per cent.

In the American Army, during the last war, there were 4,325,000 men on active service, 979 cases of small-pox, with 15 deaths, i.e., an annual admission-rate of 21 per 100,000 and an annual death-rate of 0·34 per 100,000.

During the Spanish War the admission-rate was 191 per 100,000; death-rate 55 per 100,000, and fatality-rate 28·9 per cent., and during the Civil War the figures were 800 per 100,000; 275 per 100,000; and 37·2 per cent. respectively.

Small-pox amongst the Vaccinated.—But it may be asked: "If vaccination protects against small-pox for a number of years, why should vaccinated children get the disease at all?" There are several answers:—

(1) The term vaccinated includes not only those efficiently vaccinated, but also those in whom the operation was inefficiently done. By *efficient vaccination* is meant, "*vaccination resulting in no less than four marks of total areas not less than half a square inch.*" It is quite a usual occurrence for a doctor to yield to the importunities of a mother and vaccinate her baby at one spot only, and if there is the slightest evidence that the vaccinia has taken, to issue a certificate of "successful vaccination." Such a baby's immunity cannot possibly last any appreciable time, but when the child gets small-pox during an epidemic the case is classified as one of small-pox amongst the vaccinated.

The following statistics show the variation of the extent of the immunity with the efficiency of the vaccination:—

TABLE CCII.—DR. W. GAYTON'S STATISTICS OF OVER 10,000 CASES.

	Vaccinated with four good marks		Vaccinated with imperfect marks		Vaccinated No marks visible		Not vaccinated	
Ages	0-5	All ages	0-5	All ages	0-5	All ages	0-5	All ages
Incidence ..	61	2,085	182	4,854	118	1,295	677	2,169
Deaths ..	0	62	21	455	47	352	382	938
Fatality ..	0	3 per cent.	11·5 per cent.	9 per cent.	40 per cent.	27 per cent.	56 per cent.	43 per cent.

TABLE CCIII.—MARSON'S ANALYSIS OF 13,755 CASES OF SMALLPOX.

	Percentage of deaths in each class respectively (uncorrected)		Percentage of deaths in each class respectively (corrected)	
	1836-51	1852-67	1836-51	1852-67
(1) Stated to have been vaccinated, but no cicatrix visible	25·5	40·3	21·7	39·4
(2) Having one cicatrix	9·2	14·8	7·6	13·8
(3) ,, two cicatrices	6·3	8·7	4·13	7·7
(4) ,, three ,, ...	3·6	3·7	1·8	3·0
(5) ,, four or more cicatrices..	1·1	1·9	0·9	0·9
(6) Unvaccinated	37·5	35·7	35·5	34·9

In recent years vaccination has been practised by introducing the lymph intradermally, instead of by scarification. This method, which minimises the risk of secondary infection, also produces very little scarring, and hence the size of the cicatrix is now no more any criterion of the efficacy of the vaccination, and the only test of immunity is the reaction of the individual to another vaccination.[1]

(2) Another factor which tends to make the statistics with regard to small-pox amongst vaccinated less reliable is the fact that some of those classified as vaccinated were vaccinated whilst they were already incubating small-pox for some days during an epidemic. The incubation period of vaccination is several days shorter than that of small-pox, and hence within three days of exposure to infection proper vaccination may altogether prevent an attack of the disease, and, according to Marson, the disease may be considerably modified if vaccination is effectively performed even five or six days after exposure. But vaccination later than the sixth day can neither prevent nor modify the disease. Such vaccinated cases getting small-pox (either severe or modified) go down in the records as small-pox amongst the vaccinated.

(3) There is still another consideration, viz., that even people protected by an attack of the disease itself occasionally get another attack of small-pox after a lengthy interval. The antibodies created by the disease or by vaccination gradually die out, and the chances of reinfection vary both with the interval since the attack or the vaccination, as well as with the severity of the epidemic. The longer the interval and the more massive the dose of the infection the greater the chances of the reinfection. Louis XV of France died of a second attack of small-pox, and cases are on record of people having had three attacks of the disease, although the possibility of one of those attacks having been chicken-pox must always be borne in mind.

There is still one further piece of confirmatory evidence of the protective value of vaccination, viz., **the change in the incidence-age** of small-pox from childhood in prevaccination times to adult age since the introduction of vaccination on a large scale. This is illustrated by the following statistics for England and Wales :—

[1] See J. P. Leake and S. Thomas, "The Vaccination Scar as an Index of Immunity." *Journ. Amer. Med. Assoc.*, 87, 1926, 1125.

TABLE CCIV.—DEATHS PER MILLION LIVING.

	0-5	5-10	10-15	15-25	25-45	45 upwards
1848-54	1,514	323	91	110	69	24
1855-64	789	210	69	119	88	36
1865-74	783	333	142	267	221	88
1875-84	128	63	46	82	77	34
1885-94	50	15	11	24	32	19

When calculated in percentages of the total deaths from small-pox at all ages, the mortality amongst children under 5 dropped from 67·7 per cent. in 1848-54 to 28·8 per cent. in 1890-94, and for children under 10 it dropped from 80·7 per cent. to 33·8 per cent. during the same interval. The greatest drop has occurred for the age-period 1 to 5 years, viz., from 42·6 per cent. to 11·7 per cent., as compared with the drop from 25·1 per cent. to 16·6 per cent. for the age-period 0-1. The reason is obviously the fact that as vaccination is generally performed when the infant is a few months old, there are found to be large numbers of *unvaccinated* infants under 1 year old.

The explanation for this change in age-incidence obviously lies in the fact that in the prevaccination period nearly all the adults were protected by a previous attack in childhood, and it was the child population that was unprotected. The conditions were, however, reversed by vaccination.

Moreover, as a result of recent neglect of vaccination, the percentage of children affected by epidemics is proportional to the extent to which vaccination is neglected. Thus, in Leicester and Gloucester, where vaccination is very largely neglected, the small-pox deaths among children under 10 were 71·4 per cent. and 64·5 per cent. of small-pox deaths at all ages respectively. In Warrington and Sheffield, where the child population was well vaccinated, the percentages were 25·6 and 22·6 respectively. Whilst in London and Dewsbury, which were intermediate in respect of the vaccinal condition of children, the percentage mortalities of children under 10 were also intermediate, viz., 51·8 in Dewsbury and 36·8 in London.

The following statistics, giving the percentage mortality from small-pox (according to age) in Berlin during various epidemics in pre-vaccination, partial vaccination and universal vaccination periods in Berlin, were published by H. A. Gins in 1922 :—

TABLE CCV.

Ages	1758-74	1870-72	1916-17
0-12 years ..	98·70	49·02	2·12
12-39 ,, ..	1·25	27·13	7·32
Over 39 years ..	0·07	23·85	90·56
Total	100·00	100·00	100·00

We have thus seen that the value of vaccination as a protection against small-pox has been proved both experimentally and statistically as conclusively as any fact has or can ever be proved. Nevertheless, as I stated on p. 592, this simple operation has aroused considerable opposition from a relatively small but very vociferous section of the public, and it is, therefore, right that one should give a fair hearing to the other side, and examine their arguments in an impartial manner.

Arguments of Anti-vaccinationists.—These may be put as follows :—

I. It is absurd to suppose that one disease (like cow-pox) could prevent a totally different disease (like small-pox).

II. The operation is useless, and the extinction of small-pox is not due to vaccination but to other factors.

III. The operation is harmful because : (*a*) Many deaths have occurred as the result of it ; (*b*) various diseases have been transmitted to the child in the process of vaccination.

I. As regards their first point, their contention falls to the ground, since it has been repeatedly shown experimentally that cow-pox and small-pox are identical diseases, as was believed by Jenner, who called cow-pox small-pox of the cow. This has been proved by the fact that *cow-pox* has been produced in the cow by inoculating it with matter taken from a *small-pox* patient *under conditions absolutely excluding any possible contamination of the wound with cow-pox matter.* Moreover, when the lymph from such an inoculated cow is used for human vaccination, results have been obtained which are indistinguishable from those produced by ordinary vaccination.

II. In support of their second contention the opponents of vaccination argue as follows :—

(*a*) The operation is useless because, although vaccination was made compulsory in 1871 there was, during the quinquennium 1871-75 of obligatory vaccination, a small-pox mortality of 2,610 per million as compared with one of 700 per million in the preceding quinquennium (1865-70) of optional vaccination.

(*b*) The decline of small-pox mortality after the introduction of vaccination was not due to the vaccination but to:—

(*α*) The abandonment of inoculation.
(*β*) Improvement in sanitation.

(*a*) *Is the operation useless?*—The figures given by the vaccination opponents are *true*, but the facts are *not wholly true*. It is true that vaccination became compulsory in 1871, but at the same time it is a fact that, though *compulsory*, the operation was *not properly enforced* till after the epidemic of 1871. Moreover, the 1871 epidemic could hardly have been affected by the compulsory vaccination introduced in that year. To show that in 1871, just before the epidemic, there was a considerable number of infants that had not been vaccinated, and thus dispose of the opponents' contention that "infants were thoroughly vaccinated at that time," the following calculation based on figures given by the Anti-vaccination League itself will be of interest.

The Anti-vaccination League states that the average percentage of births successfully vaccinated *by the Public Vaccinator* during 1865-69 was 48·3 per cent., and for 1870 it was 50 per cent. Hence the average for the six years 1865-70 was

$$(48{\cdot}3 \times 5 + 50)/6 = 48{\cdot}6 \text{ per cent.}$$

The League further states that 57·1 per cent. of public vaccinations in 1872 meant a total of 94 per cent. of available births. It follows, therefore, that 48·6 per cent. of public vaccinations accounts for $\dfrac{48{\cdot}6 \times 94}{57{\cdot}1} = 80$ per cent. of available births. In other words, 20 per cent. of infants were not vaccinated.

But, apart from this, it is obvious that the compulsory vaccination could not affect any of the children that were over 5 years old during the period 1871-75. Hence the figures of small-pox mortality during that period to be of any value must be divided into those for children under 5 and those over 5 years of age. When this is done, the results are as follows:—

TABLE CCVI.

	Small-pox mortality per million	
	0-5	10-15
1865-70	413	32
1871-75	937	234

In other words, whilst the mortality amongst children under 5 was increased only two and a quarter times, that amongst children 10 to 15 years old was increased ten times.

Further, even of the children under 5, it is clear that all those over one year were not affected by the Vaccination Act of 1871, and a large proportion of them must have remained unvaccinated. Therefore, for the statistics to show the full value of vaccination they should show the relative mortalities (during those two periods) of children of the vaccinated and unvaccinated. Unfortunately, this is not shown in the figures referred to. If, however, we compare the mortality figures for children under 5 with those between 10 and 15 during the two quinquennial periods, 1865-70 (five years before enforced vaccination) and 1876-80 (i.e., the first quinquennium during which all children under 5 were vaccinated), we find the following results:—

TABLE CCVII.

	0-5	10-15
1865-70	413	32
1876-80	145	52

These figures show that the *mortality of the children under* 5 who were affected by the 1871 Act *was reduced* by 65 per cent., *whilst that of the children of the age-group* 10 *to* 15 who were entirely unaffected by that Act *was increased* by 63 per cent.

Moreover, the statistics for the epidemic year 1871-2 show that the percentage of children's deaths (under 5) to total deaths from small-pox was 32·4, as compared with 55·5 in 1865-69.

The anti-vaccinationists, in their endeavour to show that vaccination was well enforced before 1871, point out that fines were imposed for evasion of the Vaccination Act of 1853. But this proves nothing, since similar persecutions took place of

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drunkards under a somewhat recent Act, when the "Black List" was instituted, but these persecutions accomplished very little at the time.

(*b*) *Is the Decline of Small-pox due to something unconnected with Vaccinaton? The Argument from New Zealand.*—To prove that it is not vaccination which is responsible for the decline of small-pox, the League absurdly points to the absence of small-pox in a comparatively unvaccinated country like New Zealand, utterly ignoring the facts that New Zealand is an entirely isolated country, the nearest land, Australia, being 1,000 miles away, and that it has a density of population only $\frac{1}{1000}$ that of London and $\frac{1}{64}$ that of the entire United Kingdom. Small-pox, therefore, has no chance of getting there or of spreading when it succeeds in finding an entry.

Argument from Japan.—Further, say the anti-vaccinationists, there is Japan, which is a thoroughly well-vaccinated country (enough vaccination having been done during twenty years preceding 1908 for every person to be vaccinated more than twice) and yet she had over 18,000 cases of small-pox with 6,000 deaths as recently as 1908. The answer to this is manifold:—

(i) The average of over two vaccinations per person is due to the fact that some have been vaccinated three or more times, whilst others have not been vaccinated at all, as is definitely stated in the official report by Professor Kitasato. For anti-vaccinationists to use such an argument is as absurd as saying that "wealth is no protection against starvation because in such a rich city as London, a considerable number of people suffer every year from starvation."

(ii) Figures published by the *Vaccination Inquirer* itself (the official organ of the Anti-vaccination League) show that out of 2·18 vaccinations per person, 50 per cent. had not taken well. This at once practically doubles the number of unvaccinated people.

(iii) Japan is in free communication with Korea, China and other places where small-pox is endemic, and, in spite of the quarantine stations which have been established, in 1899, many hundreds of cases must enter Japan in a state of small-pox incubation.

(iv) If we apply the acid test by classifying the small-pox cases according to the vaccinal condition and interval after vaccination, the results are just what one would expect. Thus:—

TABLE CCVIII.

		Cases	Deaths	Fatality-rate
1907-8	Amongst vaccinated	7,254	1,926	26·6
	,, unvaccinated ..	2,932	2,031	64·3
For 1878-82	Amongst vaccinated	3,910	455	11·63
	,, unvaccinated ..	6,090	2,190	32·73

Also in Kobe, there were :—

TABLE CCIX.

	Cases	Deaths	Fatality rate
Amongst unvaccinated (44 per cent of total)..	276	134	50·19
,, vaccinated (274 cases) within 5 years	19	—	—
,, ,, ,, over 5 years ..	255	19	7·84
Doubtful vaccination	38	6	15·79
Vaccinated during incubation period	25	3	12
Having had previous attack of small-pox ..	4	—	—

These figures, by the way, form a somewhat biting commentary on the statement quoted by the Anti-vaccination League, on the authority of an anonymous reporter in an anonymous American paper, to the effect that "in Kobe nearly 50 per cent. of the cases were fatal, and only one per cent. of the cases occurred amongst unvaccinated." No doubt the reporter was a near relative of the man who boasted that he once shot 999 birds one after another without missing one, and when asked why he did not make it 1,000, indignantly replied that he would not stoop to tell a lie for the sake of one bird!

(v) That the decline of small-pox in Japan has followed *pari passu* with introduction of vaccination is seen from the following figures :—

TABLE CCX.

	Cases	Deaths	Fatality-rate
First epidemic, 1885-7	125,875	31,972	25·48
Second ,, 1892-4	88,095	23,603	26·8
Third ,, 1896-7	52,650	15,664	29·7
Fourth ,, 1907-8 (December to August)	19,101	6,273	32·9

Vaccination was introduced in 1876, was nominally enforced since 1885, when a fine of 5 to 50 sen (i.e., 2d. to 2s.) was imposed for evasion of the law, and was more strictly enforced since 1909, when the fine for evasion increased to £1 (10 yen).

Hence, in spite of the increased virulence of the epidemics, as judged by the increased fatality-rates from 25·5 to 33 per cent., each epidemic was of shorter duration and affected fewer people than its predecessor, and notwithstanding the fact that the population had increased from 40 millions in 1885 to 50 millions in 1907. The average small-pox mortality thus fell from 800 per million in the first epidemic to 120 per million in the fourth.

Still more striking are the following figures :—

1875-84	Average	small-pox	mortality,	4,946	per million
1885-94	,,	,,	,,	138	,,
1895-1904	,,	,,	,,	37	,,

Then an apparently reasonable objection is raised to the effect that the decline in small-pox observed wherever vaccination is introduced is quite out of proportion to the amount of vaccination that has been done. But the obvious answer to this is that on first introduction of vaccination in small-pox areas the majority of the population do not require it, having already been protected by attacks of the disease itself.

Let us now examine the anti-vaccinationist's contention that the reduction of small-pox is due to the abandonment of inoculation rather than the adoption of vaccination.

(*a*) The practice of inoculation against small-pox, by putting some of the matter from a small-pox vesicle under the skin or into the mucous membrane of the nose, was known to the Chinese in A.D. 590, and Lady Mary Wortley Montagu, the famous authoress and wife of a British Ambassador at Constantinople, hearing of the custom, first introduced the method into England by practising it on her own children in 1718. This operation, which undoubtedly produced a much milder form of small-pox in the people inoculated, was on account of contagiousness made illegal in 1840, since it formed a new source of infection, and its place was taken by vaccination which confers immunity without making the person infectious to other people.

It is very difficult to say whether inoculation caused more deaths than it prevented by increasing the incidence of the disease, but that the decline in small-pox mortality is not due to the cessation of inoculation is proved by the fact that in popu-

lations like Sweden and Copenhagen, where inoculation was never practised to any large extent, the small-pox mortality suddenly fell soon after Jenner's discovery.

Thus, in the case of Copenhagen, the official statistics are as follows :—

TABLE CCXI.

	Small-pox-mortality per million	
1770-79	1,288	Average 2,000
1780-89	2,068	
1790-99	2,651	
1800-09	675	Average 265
1810-19	4	
1820-29	116	

Moreover, the Vaccination Commissioners point out that *the increase of small-pox in England during the eighteenth century occurred during the first quarter before inoculation was introduced.*.

(β) **Sanitation versus Vaccination.**—As an alternative to the cessation of inoculation, it has been suggested that the fall in small-pox mortality is due to improved sanitation. That this is not so is proved by many facts. In the first instance, small-pox began to decline in the beginning of the nineteenth century, whilst the important Public Health Act was passed only in 1875. But apart from that, it is difficult to see what exactly the opponents of vaccination understand by sanitation. It cannot mean the same sort of sanitation as is responsible for the disappearance of typhus (abolition of dirt and vermin), of typhoid fever (improved water supply), or for the decline of scarlet fever, measles, &c., since :—

(i) Immediately after the introduction of the Public Health Act there was a sudden change from 6·1 to 3·5 in the incidence of typhus during the age-period 0—5, with no further change since then, but in the case of small-pox the change was sudden after the introduction of vaccination, and had been going on gradually since then until the time that vaccination began to be neglected. Thus, the percentages of deaths under 5 years (to deaths at all ages) for four successive quinquennia in the case of typhus and small-pox have been :—

TABLE CCXII.

	1871-75	1876-80	1881-85	1886-90
Typhus ..	6·4	6·1	3·5	3·4
Small-pox ..	16·0	13·0	12·0	1·8

(ii) Typhoid fever mortality is twice as high in New Zealand—where small-pox is absent—as it is in London. Indeed, the cause of the reduction of typhoid fever has been determined with certainty, viz., purification of water supply, and while purification of water supply in any locality eliminates with certainty typhoid fever, it does not influence the small-pox mortality in the slightest degree.

(iii) In India small-pox is rampant, but typhoid and scarlet fever are unknown. Also the fact that properly vaccinated doctors and nurses in attendance on small-pox patients do not contract small-pox, whilst some of those in attendance on diphtheria and scarlet fever do contract these diseases to a considerable extent (unless, in most cases, they previously had these diseases, or, in the case of diphtheria, have been properly immunised by means of toxin-antitoxin), is proof positive that small-pox is not due to the same causes as are responsible for the other infectious diseases, and can certainly be controlled by timely efficient vaccination. Moreover, the relative mortality of the revaccinated British Army in India is only one-sixth that of the general population, whilst its typhoid mortality is forty-three times as high. On the other hand, in Ireland typhus is still present, whilst small-pox is practically extinct.

(iv) Measles mortality has declined from 398 per million in 1850 to 138 per million in 1923, whilst small-pox mortality has gone down from 262 to zero per million.

As an offset to the table comparing the behaviour of vaccinated with that of unvaccinated places in Germany and Switzerland (see Table CXCVI, p. 597), the anti-vaccinationists quote Sheffield, which, though well vaccinated, had 192·4 cases per 10,000 in 1887-88, whilst unvaccinated Leicester in 1892-93 had only 19·3 cases per 10,000. The obvious answer is, that whilst Table CXCV

compares two localities during the same period of years, the figures for Sheffield and Leicester are for two separate periods.

W. R. Macdonell[1] found the **correlation coefficient** between vaccination and recovery from smallpox to be 0·656 ± 0·009, and whilst this high correlationship does not in itself prove any causal relationship between the two, it affords very valuable confirmatory evidence.

III. Risks of Vaccination.

(*a*) The anti-vaccinationists say that many children die as the result of vaccination. The figures, including deaths after vaccination, where the death had nothing to do with the operation, work out at one death per 25,000 vaccinations for England and Wales. In Germany the mortality from vaccination accidents is about one in 100,000. But even this small mortality is avoidable if glycerinated calf-lymph is employed under aseptic precautions. The most important complication of vaccination is erysipelas, and yet during 1883-87 there was an increase of 41·5 per cent. of deaths from erysipelas in unvaccinated Leicester, compared with a diminution of 16·7 per cent. over the whole of England and Wales. But, granting that an accident *may rarely* happen, "Is," to quote Walpole, "one child lost an argument against millions saved?" In the words of Seneca, "Sometimes a vessel perishes in harbour; but what do you think happens on the open sea?" (Epistle xiv). In 1911 there were 18,000 people killed or injured in the streets of London out of 1,268 million passengers carried by trams or 'buses, i.e., fifteen people killed per million passengers, a mortality similar to that from vaccination, but this would hardly be an argument for the abolition of vehicular traffic in the streets. Further, would any sane person condemn eating because cases of ptomaine poisoning occasionally arise?

(*b*) The question of transmission of disease by vaccination has been raised. That such transmission was possible with arm-to-arm vaccination (i.e., vaccination by means of lymph taken from the arm of a vaccinated person) cannot be denied, although the frequency of such an occurrence has no doubt been greatly exaggerated. The use of glycerinated *calf-lymph*, however, under the Vaccination Act of 1898, has obviated all possibility of such accidents, since it has been proved that no germs, other than those of cow-pox, are present in such lymph.

The English Vaccination Law.—It is unnecessary to consider the various legislative Acts that have been passed regarding

[1] *Biometrika*, 1901-02, vol.

vaccination since 1840. Suffice it to say that under the Vaccination Act of 1898, which is now in force, compulsory vaccination has been virtually abolished, since under its "Conscience Clause" no child need be vaccinated if the parent satisfies a magistrate (i.e., any J.P.) that he has a conscientious objection to vaccination on the ground that it would injure the child's health. As there are many J.P.'s who are either ignorantly indifferent regarding vaccination, or are actual anti-vaccinationists, it is obvious that exemption from vaccination can be procured without much trouble.

Those who fail to avail themselves of the Conscience Clause, or to satisfy a magistrate about their claim for exemption, must have their children vaccinated before they reach the age of six months, unless a medical practitioner certifies the child to be in a bad state of health, when the operation is postponed until the child is well again. Since the passing of the Vaccination Act of 1898, the number of vaccinations in England and Wales dropped from 68 per cent. (in 1893-97) to 40 per cent. (in 1922) of the total number of births.

The Act as it stands now is, therefore, from the point of view of the prevention of small-pox, practically useless, since the untiring activities of anti-vaccination propagandists easily influence the ignorant and the gullible portion of the public, whilst the more cultured and scientifically-minded section of the population hardly require an Act of Parliament to induce them to have their children vaccinated. It follows, therefore, that either the Act should be repealed, and thus all semblance of compulsion, where there is no compulsion, be removed, or that the Conscience Clause be eliminated. Believing, as I do, in the harmlessness of the operation, and its immense protective value against a most horrible disease, I am of opinion that the second alternative is the one which should be adopted. There are, however, some (e.g., Dr. Killick Millard, M.O.H. for Leicester) who believe that, although vaccination does all that it claims to do, the operation is not necessary at the present day when small-pox is practically extinct, and should only be adopted when any outbreak of the disease occurs in that locality. This might be a reasonable position to take up, except that: (1) When any serious outbreak occurs it is bound to attack and kill a considerable number of unprotected persons; (2) it takes some time to get vaccinated during an epidemic, and many might be vaccinated whilst they are in an advanced stage of incubating the disease, when the operation

would fail to protect them. It is well to remember the wise counsel which Seneca makes the nurse give Queen Clytemnestra ("Agamemnon," line 145): *Caeca est temeritas quae petit casum ducem.* ("Blind is he and rash who follows chance.")

There is still another section of people who say, "Why have compulsory vaccination? Let those who believe in it protect themselves, whilst those who do not believe in it, let them suffer for their folly." This standpoint is unjustifiable for several reasons. In the first instance, it is not always the case that only "the soul that sinneth, it shall die," but a case of small-pox occurring in an unvaccinated person might become a nuisance, if not a danger, to others who had been vaccinated a long time ago.

In the author's opinion, the position is as illogical as that of allowing rows of houses to be built of highly inflammatory material in a street where the other houses are fireproof. Secondly, it is unfair to expect the ratepayers to maintain small-pox hospitals for the treatment of patients who contracted the disease entirely through their own folly and obstinacy. I believe that in a well-vaccinated country like Germany there are only a few beds reserved for small-pox cases—and they are in the wards of general hospitals. The small-pox epidemic in 1901-03, with its 10,000 cases and 1,600 deaths in London, cost the Metropolitan Asylums Board £500,000 for isolation and treatment, and a big epidemic would, of course, cost much more. However, I am not concerned with the legislative part of the subject, and if I have succeeded in convincing the reader of the complete immunising value of vaccination and revaccination, I am satisfied to leave the controversy regarding *compulsory* vaccination in the hands of other and more competent persons.

The Hygiene of Vaccination.—The best time to vaccinate a baby is at about two to three months, when it is neither too young to have its nutrition appreciably disturbed temporarily, nor too old for it to scratch and infect the sore places.

If the child is in bad health, or there is a case of infectious disease or erysipelas in the house, the operation should be postponed till the child is better or the conditions have changed. If, however, there is small-pox about, vaccination must be done at once, whether the health is good or bad and whether the surroundings are favourable or otherwise.

The vaccination may be done on the arm or on the leg. In any case the place must be kept aseptically covered and dry. The vaccinated part must therefore not be bathed.

Tuberculosis.

A child born to parents, one or both of whom are tuberculous, is extremely liable to infection, because not only is its natural resistance low, but its chances of infection through being constantly in the company of its parents are very great. Separation of such a baby from its mother is, as a rule, attended with good results as far as the danger of infection is concerned, but it has the obvious drawback that not only is it deprived of its mother's milk, but also of other maternal care at a time when it is most needed.

Calmette, Guérin and their co-workers,[1] experimenting on calves and monkeys, found that when the bovine tubercle bacillus is attenuated in a special way (i.e., by cultivating for 230 successive generations on pure ox-gall, which is very rich in lipoids), it completely lost its virulence without being deprived of its immunising power, so that a culture thereof injected intravenously into a calf or monkey rendered it subsequently insusceptible to intravenous injection of 5 mg. of virulent bacilli, although non-vaccinated animals succumbed to such a dose in two months. In other words, by such special cultivation the tubercle bacillus becomes non-tuberculogenic, although still capable of producing antibodies. Monkeys, though naturally very susceptible to tuberculosis, remained immune after vaccination, even if placed side by side for a long period with infected monkeys, although unprotected monkeys living in such close cohabitation with infected monkeys caught the disease.

In view of these successful results in animals, Calmette and his collaborators felt justified in trying the effect of such immunisation on new-born infants, especially those brought up by tuberculous mothers. Statistics show that the mortality from tuberculosis during the first year of life among such infants is not less than 19 per cent. of the total infant mortality among them. In Paris the proportion seems to be 32·6 per cent. In view of the absorptive power of the small intestine in respect of microbes being very considerable in newly-born infants, Calmette gave three doses of 10 mg. of B.C.G. (Bacille Calmette Guérin), mixed with a little milk, by the mouth to new-born infants during the first ten days of life.

Between July 1, 1924, and January 1, 1926, immunisation with B.C.G. emulsion, supplied by the Pasteur Institute, Paris, was done in the case of 4,183 new-born infants, of whom 1,317

[1] *Annales de l'Institut Pasteur*, xxxiv, 1920; *ibid.*, February, 1926.

have been individually controlled throughout that period. The following table represents the results :—

TABLE CCXIII.

	Total number immunised	Number alive Jan. 1, 1926			Number dead Jan. 1, 1926		
		In contact with open tuberculosis	Not in contact with open tuberculosis	Total	In contact with open tuberculosis	Not in contact with open tuberculosis	Total
Infants immunised between July 1, 1924, and Dec. 31, 1924	564	231	288	519	43	2	45
Infants immunised between Jan. 1, 1925, and July 1, 1925	753	355	336	691	53	9	62
Total	1,317	586	624	1,210	96	11	107

Hence, of the 1,317 immunised infants, 586 remained in contact with bacilliferous parents, relatives, or strangers, for periods varying from between six to eighteen months; 107 died. Of these, 11 only died of tuberculosis, giving a total tuberculosis death-rate amongst the 1,317 immunised of 7 per 1,000, and amongst the 586 in contact with open tuberculosis of 18 per 1,000. As the average tuberculosis death-rate amongst non-immunised infants of tuberculous parents is probably about 250 per 1,000, it would seem that immunisation saves at least 93 per cent. of infants who would otherwise succumb to tuberculosis during the first year of life. These immunisations have never met with any untoward results and are therefore perfectly safe.

At the Pasteur Institute, in Paris, the method is now carried out in a routine manner, and careful records are kept of all cases. The vaccine does not keep longer than ten days and hence cannot be kept in stock, but is sent out by the Institute on the receipt of a telegraphic birth notification from a doctor. The dose, contained in an ampoule, is given by the mouth in a small teaspoonful of milk at body temperature half an hour before a feed. The first dose is given as soon as possible after birth, and is repeated two and four days later (i.e., on the third and fifth days). Less than three doses is not sufficient. It is, in addition, advisable to separate the immunised infant from massive infection.

Calmette claims that this immunisation by the mouth is perfectly harmless and gives protection for about three years. The method is, of course, as yet in its experimental stage, but should it be substantiated, it is possible that within two or three generations tuberculosis might be practically extinguished, especially when combined with similar immunisation of cattle, as carried out by Nathan Raw in this country. Calmette recommends re-immunisation at the end of the first and third years.

The only weak link in this otherwise strong chain of evidence in favour of the immunising value of B.C.G. is the figure relating to the tuberculosis-mortality of non-immunised infants brought up in a tuberculosis environment. Calmette assesses it as 250 per 1,000, but he does not give details how he arrived at such an estimate. Exact figures recently collected in Denmark[1] give a tuberculosis-mortality among such infants of between 49 and 96 per 1,000. But even compared with such lower figures, Calmette's results are sufficiently striking.

In the case of older children, until Calmette's method has been thoroughly tested and confirmed, all those coming from tuberculous stock, or living in houses in which are cases of tuberculosis, should, as far as possible, be sent to open-air schools and be specially cared for in the way of sufficient nourishing and digestible food. When an open-air school is impracticable, the class-room should be a thoroughly well-ventilated one.

The "Œuvre Grancher," founded in 1905 by the man whose name it bears, aims at removing infected children, 3 to 10 years old, from the care of their tuberculous parents and their infected homes, and to board them out in the country. Of the 2,300 children thus boarded out between 1905 and 1920, only seven showed tuberculosis, and out of these two died of tuberculous meningitis within a very short while of removal from home. This gives a morbidity-rate of 3 per 1,000, and a tuberculous mortality-rate of 1 per 1,000. In those not so isolated the morbidity-rate is said to have been 600 per 1,000, and the mortality-rate 400 per 1,000! But here, again, we have no clear evidence that the mortality of the non-isolated children was really so high.

The importance of environment in infant tuberculosis is well illustrated by the experience of von Bernheim in the case of three tuberculous mothers with twins. In each case one

[1] R. Kjer-Petersen and J. Ostenfeld, *Ugeskrift for Læger*, March 31, 1927, p. 257 (cited in an editorial in *Brit. Med. Journ.*, May 7, 1927, p. 845).

was left to be nursed at home by a wet-nurse, and the other was taken to the country and artificially fed. The three infants left at home died in spite of breast-feeding, and the other three remained well.

Rheumatic Infection.

There is very little doubt that rheumatic manifestations in children, i.e., rheumatic fever, St. Vitus' dance, recurrent sore throats, heart disease and growing pains are of microbic origin. According to some authorities, different kinds of bacteria circulating in the blood will, in a predisposed child, bring about an attack of the disease. This view has, however, failed to secure general support, and it seems certain that rheumatic fever is a *specific* microbic disease caused by one specific organism—although the exact identity of that organism cannot be said to be known with certainty. Poynton and Paine have isolated a streptococcus from the valves of the hearts in rheumatic endocarditis cases, and although there is very strong evidence in favour of their view that this is the true *Streptococcus rheumaticus*, many bacteriologists fail to share that view. The experimental evidence in support of Poynton and Paine's contention is that cultures of the organism obtained from the joints, tonsils or heart of rheumatic cases, inoculated into animals produce typical symptoms of the disease, viz., inflammation of the joints as well as of the cardiac valves, and when injected into the brain produce symptoms of chorea or St. Vitus' dance. Moreover, the organism is said to have been isolated from the cerebro-spinal fluid of chorea patients.

There are some people who even believe that rheumatic fever is infectious from patient to patient, but I do not believe there is any very substantial evidence in favour of this view.

Predisposing Causes of Rheumatic Fever.—In addition to the microbic origin of the disease, there are certain factors which predispose the child to the invasion of the rheumatic micro-organism. These factors are:—

(1) *Heredity.*—As a rule rheumatism runs in families, but whether the disease is definitely hereditary, or whether members of the same family get the disease because they live under the same environmental conditions with respect to housing, social position, &c., is not known with absolute certainty. Probably it is truly hereditary.

(2) *Environment.*

(i) *Age.*—The disease being microbic its incidence, as might be expected, is greatest in childhood, and the school age is the most favourable period for its occurrence.

(ii) *Social Status.*—It occurs more frequently in the poorer classes and is, therefore, more prevalent in State elementary schools than in private schools, but the degree of prevalence is not proportional to the degree of poverty.

(iii) *Climate.*—The disease is more prevalent in the countries within the temperate zone, and during the cold, wet months (October to March).

(iv) *Housing Conditions.*—House dampness has also been found to favour the onset of the disease, and out of 196 cases of rheumatism investigated, 122 have been found to come from damp houses (i.e., 62·2 per cent.).

(v) *Tonsillar Infection.*—The nasopharynx, and especially the tonsils, are believed to afford the most common portal of entry for the causative organism, but the evidence is not conclusive; especially is this the case regarding the association between simple enlargement of tonsils and rheumatism. Thus, Sir George Newman quotes Dr. Ball to the effect that out of 662 rheumatic cases examined, 102 (i.e., 15·4 per cent.) had large tonsils, while out of 1,913 non-rheumatic cases, 390 had large tonsils, i.e., 15·1 per cent. In other words, the incidence of enlarged tonsils was identical in both rheumatic and non-rheumatic cases.

Importance of Heart Disease in Children.—The importance of "growing pains" and of other rheumatic manifestations in childhood will be realised from the fact that these rheumatic affections stand at the head of the list of causes of absence from school. Sir George Newman reports that out of about 707,000 children who underwent routine inspection in 1922 in certain representative areas in England and Wales, about 5,800 suffered from heart disease, i.e., a proportion of about 8 per 1,000. The New York Association for the Prevention of Heart Disease, in an investigation of 130,000 children, found the proportion to

be 7 per 1,000, although various other investigators give much higher proportions.

Dr. Carey Coombs believes that two-thirds of all rheumatic heart disease in towns occur during the school age, and Dr. R. A. Atkins estimates that 12,000 to 20,000 deaths occur in England and Wales every year from rheumatic heart disease contracted in childhood.

It is to be remembered that some 80 per cent. of all cases of heart disease in children are associated with rheumatic fever, growing pains, chorea, or recurrent sore throats, and that infection of the heart with the rheumatic poison usually means structural damage and consequent weakness of the heart-muscle wall or valves, resulting in impairment of the circulation. These facts will make the reader realise that the rheumatic infection is not only bound to interfere with the child's nutrition, but is also likely to lead to heart failure later on.

Prevention.—From what I have said about the causes and consequences of rheumatic infection, it follows that the prevention of rheumatic manifestations, and consequently also the prevention of heart disease, will be best achieved by paying special attention to children with hereditary predisposition to these infections. Such children must be specially guarded against damp and sudden chilling—such as cold bathing. Oral and nasal hygiene—in the way of preventing or remedying dental caries, septic tonsils, nasal catarrh, &c.—should be carried out in all children, and attention should be paid to the general nutrition of the child, so as to raise its resisting powers. Should the slightest manifestation of the disease show itself, the physician must carefully examine the heart, and if there is any suspicion of cardiac involvement, the child must be kept at rest in bed for a prolonged period and then sent to an open-air school.

The Need of Sanatoria or Convalescent Homes for Rheumatic Children.—The rheumatic child is as much in need of a special convalescent institution as the tuberculous or pretuberculous individual, and the public must realise the enormous damage that heart disease—which is definitely a preventable disease—has on the economic and social life of the community. For economic reasons the State will not, under the present financial conditions of the country, grant any special aids in this direction in the same way as it provides institutions for the treatment of tuberculosis. But all State-aided institutions have had their origin in establishments initiated as the result of campaigns instituted

by private philanthropic efforts, and it is for the Press of this country to undertake a similar campaign on behalf of the rheumatic child. In 1919, Mr. Charles Lee gave the Edgar Lee House, in Willesden, to the Invalid Children's Aid Association in memory of his only son, who fell in the war. This house has room for twenty-two rheumatic boys, where they can remain for as long as a year or longer, and have the necessary rest, medical supervision, as well as the necessary schooling, towards which the parents contribute according to their means. This home is doing good work, and similar institutions ought to be established throughout the country.

Since the above was written, the Metropolitan Asylums Board have, on April 9, 1927, decided to provide an extension of 350 beds for cases of rheumatism at Queen Mary's Hospital, Carshalton.

Special Cardiac Classes. — Children suffering from heart affections when attending ordinary schools are handicapped in their competition with their normal schoolmates to an extent varying with the degree of their cardiac defect. Not only are they, as stated above, frequently absent from school, but they are not able to take part in athletic games or to climb stairs. Special classes have, therefore, been established in America for the needs of such children. They are placed on the ground or first floor of the school building, and the physical and mental work is specially adjusted to their cardiac efficiency, as estimated by special tests, such as exercise tolerance, &c. The chief objection against such classes is that of the parents who consider that a special stigma is attached to a child who is segregated from the normal school children. The results, however, where these classes have been established are so good as to outweigh this or any other objection. The number of days of absence from school by such children has been reduced by about 75 per cent., and the gain in weight has been at the rate of about 1 lb. a month per child.

Effects of Tonsillectomy.—Since an attack of rheumatism can usually be dated from a sore throat, and the *Streptococcus rheumaticus* has been isolated from the tonsils of rheumatic patients, it would appear that tonsillectomy should prevent a first attack of rheumatism. Unfortunately, statistics of different observers do not agree. Some people regard tonsillectomy as a prophylactic, whilst others find it has no influence on the incidence of the disease. Thus, statistics published by Hunt and Osman, giving the results of their investigation by the "follow-up"

method, i.e., following up for a period of years a number of rheumatic cases and seeing what has happened in the way of recurrence to cases that have had the tonsils removed and to those that have not had their tonsils removed, show that the recurrence in tonsillectomised cases is 53 per cent. as against only 33 per cent. in non-tonsillectomised cases. On the other hand, Ingerman and Wilson found the percentage of recurrences after tonsillectomy to be 76 per cent. as against 80 per cent. in non-tonsillectomised cases. Reginald Miller, however, rightly contends that the follow-up method is unreliable, and that to obtain a true idea of the effect of tonsillectomy one should ascertain the subsequent history of cases with *prodromal symptoms* of rheumatism, or alternately to study the manifestations of rheumatism in tonsillectomised children who had never had a previous attack of the disease. He found that: In 27 cases of post-tonsillectomy rheumatism, 17 (= 63 per cent.) showed chorea (i.e., St. Vitus' dance), and only 1 each (= 4 per cent.) showed inflammation of the joints or affection of the heart, whilst in 45 cases of pre-tonsillectomy rheumatism 13 (= 29 per cent.) showed chorea, and 17 each (= 38 per cent.) showed inflammation of the joints or heart affection.

In other words, the heart and joint affections are definitely diminished by tonsillectomy, whilst the incidence of chorea seems to be increased by the operation. Miller, however, is of opinion that this increase is apparent only, and is in most part due to the fact that the other serious types of rheumatism are greatly diminished.

Taking all the evidence into consideration, it is, in the author's opinion, impossible to decide how far indiscriminate tonsillectomy will diminish the incidence of rheumatic infection. Although the removal of septic tonsils should certainly be done in suitable cases, it is probable that oro-nasal hygiene (painting the tonsils daily with Mandl's paint containing iodine, and thorough nose blowing), fresh air, good food, proper clothing, avoidance of damp and general hygiene will do more for the prevention of rheumatism than wholesale removal of all large tonsils.

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CHAPTER XXV

THE PHYSICAL AND MENTAL GROWTH OF THE CHILD

"A child is a man in a small letter, yet the best copy of Adam; and he is happy whose small practice in the world can only write his characteristics. He is Nature's fresh picture newly drawn in oil, which time and much handling dims and defaces. His soul is yet a white paper unscribbled with observations of the world, wherewith at length it becomes a blurred note-book."—BISHOP EARLE.

Physical Growth. — As has already been pointed out in Chapter X, p. 296, the child differs from the adult not only in size and weight but also in regard to the proportional sizes of the various parts of its body (fig. 76). This fundamental fact has an important influence on the weight of the body at different ages. Were the bodily proportions to remain constant throughout the period of growth, it would at any moment be easy to calculate the theoretical weight of any individual from a knowledge of any one of his linear dimensions, such as height and *vice versa*, provided his weight, as well as the size of that linear dimension at birth, is known, and the specific gravity of the body did not alter through any change in its gross chemical composition. For, elementary geometry teaches us that the volumes of two similar figures are proportional to the cubes of their linear dimensions, and, therefore, if the specific gravity as well as the bodily proportions of the adult were to be the same as in the infant, we would expect their relative weights to be proportional to the cubes of their lengths. In other words, we would expect their weights to be in the proportions of $170^3 : 50^3$ or as $3{\cdot}4^3 : 1$; i.e., the adult should weigh forty times his own birth weight. Similarly we would expect the surface area of the adult's body to be $3{\cdot}4^2 : 1$, i.e., about twelve times his own birth surface. As a matter of fact, the adult's weight is no more than about twenty times his birth weight, and his surface not more than about nine times his birth surface. Hence we see that as the result of the differences in the rates of growth in the various parts of the body, the increase in weight is no more than about

half, and that of his surface no more than three-quarters, of what it should be if all the parts of the body grew uniformly.

Even such early philosophers as Pliny and Aristotle had fairly accurate ideas regarding the growth of children, but the famous Belgian astronomer and mathematician, Lambert Adolphe Jacques Quetelet, was the first to study the subject on scientific lines.[1] He showed that although adults of the same age are not necessarily of the same height, nevertheless the variations, or deviations in height from the mean, followed the law of the normal frequency curve (see p. 90). Geissler and Uhlitzsch in 1888 and Riedel in 1913 found the same to hold good in the case of school children. Quetelet's work was followed by that of Franz Liharzik[2] who made many measurements of the different parts of the body from birth onwards, by means of which he thought he could, with the aid of geometry, formulate the laws of growth. In 1877 appeared the work of Bowditch on 14,000 boys and 11,000 girls in Boston, and in the same year Pagliani published the results of his investigations on 1,048 boys and 968 girls in Turin. The Report of the Anthropological Committee of the British Association on over 40,000 people appeared in 1885. Since then numerous investigators such as Hertel in Denmark, Axel Key in Sweden, Schmid-Monard in Germany, Erismann in Russia, Shrubsall, Tuxford and Glegg, as well as Arthur Greenwood in England — to mention a few only — interested themselves in the subject of growth.

The comprehensive table on pp. 628 and 629 giving the weights when naked, heights and other important measurements of 1,400 *healthy* children from birth up to the age of 14 years is given by Bernard Myers,[3] who carried out his investigations at the Children's Clinic in Marylebone Road.

From these measurements we learn the following facts :—

A. Absolute Measurements.

(*a*) **Weight.**

(i) The *average birth weight* is about 3·2 kg. (about 7 lb.), being slightly higher in males than in females.

(ii) The birth weight: (α) doubles itself between the 5th and 6th month; (β) trebles itself between the 12th and 15th month; (γ) quadruples itself between 2 and 2½ years; (δ) quintuples itself at about 5 years. In other words the

[1] "Sur l'homme et le développement de ses facultés," Bruxelles, 1835.
[2] "Das Gesetz des menschlichen Wachstum," Wien, 1858.
[3] *Brit. Journ. Ch. Dis.*, xxiii, 1926, p. 87.

rate of growth rapidly diminishes with increasing age. During the first year the increase in weight is 200 per cent.; during the second it is no more than 20 per cent.; and thereafter it is no more than about 10 per cent. annually.

(*b*) **Length.**

(i) *The average birth length* is about 50 cm. (20 in.), being slightly greater in males than in females.

(ii) The birth length: (α) increases by about 40 per cent. during the 1st year; (β) doubles itself between the 4th and 5th year; (γ) trebles itself between 13th and 14th year.

(*c*) **Sitting height** (i.e., the distance between the seat in which the child sits upright and the top of his head).

(i) *The average birth sitting height*, as found in an investigation carried out under the auspices of the Medical Research Council, in Glasgow, Edinburgh and Dundee, is approximately 13·5 in. (about 34 cm.).

(ii) The birth sitting height; (α) increases by about 6 cm. (i.e., about $2\frac{1}{2}$ in.) or by about 17 per cent. at the age of 6 months; (β) increases by about 10 cm. (or 4 in.), i.e., by about 30 per cent. at the age of 1 year; (γ) increases by 17 cm., i.e., by 50 per cent., at the age of about 2 years; (δ) nearly doubles itself at 13 to 14 years.

(*d*) **Head.**

(i) *Circumference.*—(α) At birth, it is about 14 in. (35 cm.) being larger in males than in females; (β) at 6 months, it is about $17\frac{1}{2}$ in. (43 cm.); (γ) at 1 year, it is about 18 in. (45 cm.); (δ) at 13 to 14 years it is about 21 in. (53 cm.). At all ages the female head is, as a rule, smaller than that of the male.

(ii) *The Sutures.*—At birth, the cranial bones are united together by membranous sutures. These sutures become ossified by the 6th month except at the anterior fontanelle. The posterior fontanelle closes by the end of the 2nd month, whilst the anterior fontanelle normally closes by the 18th month.

(iii) *Cephalic index*, i.e., the percentage ratio that the maximum width of the skull bears to its maximum length $\left(\frac{\text{transverse diameter}}{\text{antero-posterior diameter}} \times 100\right)$. An index less than 79 makes the skull dolichocephalic, or long-headed. An index between 79 and 81 makes the skull mesocephalic; whilst one of over 81 makes it brachycephalic or broad-headed. The measurements of a baby's skull taken after

TABLE CCXIIIA.—SHOWING THE MEASUREMENTS OF THE HEAD, CHEST, AT DIFFERENT AGE-PERIODS

Age-period	Head: Circumference (centimetres).		Head: Transverse diameter (centimetres).		Head: Antero-posterior diameter (centimetres).		Chest: Circumference at nipples (centimetres)	
	Male.	Female.	Male.	Female	Male.	Female	Male	Female
0 to 1 month	36·0 40·0-31·3	35·0 39·0-30·0	9·8 11·1-8·7	9·6 10·3-8·9	12·2 13·4-11·2	11·9 13·3-11·0	34·0 39·6-30·5	33·7 37·5-27·0
1 to 2 months	37·9 41·0-34·8	37·6 43·2-34·5	10·3 11·2-9·5	10·1 11·5-9·2	12·7 14·3-11·8	12·8 14·4-11·5	37·1 41·3-30·5	36·7 43·5-29·0
2 to 3 months	39·0 42·0-32·5	38·4 41·5-35·5	10·7 12·0-10·0	10·5 11·3-10·0	13·5 15·2-12·1	13·3 14·2-12·5	38·5 43·0-34·9	37·1 41·5-34·0
3 to 4 months	40·0 46·0-36·3	36·2 41·0-33·0	10·7 12·0-10·1	10·7 12·0-10·2	13·8 16·1-12·4	13·3 14·4-13·2	40·2 44·0-37·6	39·4 41·5-37·0
4 to 5 months	41·7 43·4-41·0	40·8 42·9-37·5	11·1 11·6-10·7	11·1 11·9-10·2	14·5 15·1-13·7	14·0 14·6-13·5	41·3 43·0-39·5	40·8 43·5-38·5
5 to 6 months	41·9 43·8-39·0	41·1 42·7-39·0	11·8 12·7-11·2	11·2 11·9-10·3	15·1 16·0-13·5	14·2 14·9-13·9	42·3 45·0-35·5	40·0 44·5-36·7
6 to 9 months	43·5 45·2-39·3	44·0 45·7-42·5	12·2 12·8-10·5	11·8 12·7-11·4	15·0 16·1-13·4	15·1 16·4-14·2	42·4 47·0-34·5	44·9 49·5-39·3
9 to 12 months	45·5 47·7-42·0	44·7 51·0-41·5	12·5 13·2-11·6	12·2 13·7-11·0	15·4 16·5-13·5	15·5 17·4-13·9	45·1 49·5-41·0	45·1 54·0-40·0
12 to 15 months	46·5 49·0-43·0	44·9 45·5-44·4	12·5 14·0-11·8	12·5 13·0-12·2	15·9 17·2-14·3	15·2 16·6-13·0	46·0 50·5-42·5	43·7 45·5-40·5
15 to 18 months	48·1 53·5-45·5	46·2 50·3-44·3	13·1 14·6-12·5	12·5 14·4-11·7	16·5 18·3-15·6	16·1 17·2-15·2	46·9 51·0-44·0	46·7 51·2-41·5
18 to 21 months	47·7 51·0-46·0	46·3 48·6-40·5	12·8 14·2-11·6	12·6 13·5-11·8	16·3 17·5-14·3	15·9 17·3-14·0	48·5 56·5-44·3	46·5 50·0-42·5
21 to 24 months	47·2 51·1-43·0	48·3 49·5-45·5	12·8 13·8-11·9	13·1 14·0-12·2	16·3 17·5-15·4	16·6 17·4-15·2	47·7 52·0-39·5	47·0 50·5-45·2
2 to 2½ years	49·1 53·1-45·0	47·5 52·3-43·0	13·2 14·4-12·3	12·8 14·5-11·1	16·8 19·8-15·2	16·2 17·4-14·6	50·1 53·0-48·0	48·4 56·0-40·5
2½ to 3 years	48·6 51·2-47·5	47·7 50·7-45·7	13·2 14·2-12·4	13·1 14·2-12·2	16·8 17·7-15·7	16·3 17·5-15·2	50·0 53·0-43·5	49·1 53·1-45·5
3 to 4 years	49·8 52·0-48·0	48·7 52·7-41·8	13·6 15·3-12·5	13·2 14·1-11·4	17·4 18·4-16·1	16·7 18·1-15·6	52·7 56·7-48·5	49·8 53·5-44·0
4 to 5 years	49·5 53·1-45·2	49·3 52·0-45·0	13·4 14·5-12·4	13·3 14·7-12·5	17·3 18·3-15·6	16·9 18·5-15·1	52·7 61·2-48·0	52·2 57·0-43·0
5 to 6 years	50·6 53·0-48·0	49·5 52·7-45·2	13·6 14·6-12·8	13·4 14·5-12·4	17·6 18·5-16·5	17·1 18·0-16·3	54·3 62·0-45·5	53·2 61·0-49·0
6 to 7 years	50·6 54·5-45·5	50·2 52·9-47·7	13·6 15·7-12·2	13·6 16·0-12·2	17·7 19·0-16·4	17·5 18·2-15·6	55·5 60·0-50·0	54·6 59·0-46·5
7 to 8 years	51·9 54·5-48·3	50·5 52·2-48·0	14·0 15·5-12·9	13·7 14·8-12·9	18·0 19·5-16·6	17·5 19·1-16·8	57·5 64·1-50·0	56·0 60·5-50·0
8 to 9 years	51·9 54·0-48·0	50·6 54·8-47·9	14·1 15·9-12·7	13·5 14·3-12·8	18·0 19·2-15·8	17·5 18·5-16·4	59·6 65·0-56·5	57·0 62·5-51·5
9 to 10 years	51·4 53·7-49·8	51·2 53·0-46·0	14·0 15·2-13·0	13·4 14·5-11·0	17·7 18·8-16·5	17·4 18·6-16·3	60·6 64·1-53·5	59·8 65·2-53·0
10 to 11 years	52·3 55·5-49·5	51·6 54·3-48·0	14·0 15·2-13·0	14·0 18·5-12·7	18·4 19·2-17·4	17·4 19·0-14·8	63·0 69·5-55·2	60·0 68·5-53·3
11 to 12 years	52·9 55·1-49·2	51·6 55·2-48·4	14·2 15·5-13·6	13·7 15·0-12·5	18·4 20·1-17·2	17·6 18·7-16·0	63·2 68·5-58·0	62·2 68·5-57·0
12 to 13 years	52·9 55·0-50·6	51·9 55·0-49·3	14·3 15·4-13·7	14·1 17·5-13·2	18·2 20·4-16·9	17·8 18·8-16·9	67·8 80·0-61·0	63·8 71·0-58·0
13 to 14 years	53·6 57·0-51·7	52·0 56·2-49·0	14·2 15·0-13·9	13·7 14·8-12·8	18·2 19·0-17·6	17·7 18·9-16·9	67·1 71·0-64·0	66·7 73·2-53·5

Note.—The first measurement or weight given is the average one for the age-period

Abdomen, Intercristal Diameter, Height and Weight for both Sexes (Children's Clinic).

Abdomen Circumference at umbilicus (centimetres)		Intercristal diameter (centimetres).		Height Standing (centimetres)		Height. Sitting (centimetres).		Nett weight in lb. and oz.	
Male.	Female.	Male	Female	Male	Female	Male	Female.	Male	Female.
33 7 41 0-28 5	33 4 40 0 27 3	8·9 11·2-7·6	8·6 10 9-6 7	51 2 59·8 45 7	50 8 58 4-45 7	—	—	7 lb 7 oz. lb oz lb oz 10 15 5 6	7 lb 5 oz. lb oz. lb oz 11 6-5 2
37·6 46 0 23 5	36 0 45 5 28 0	9·7 12 3 7 9	9·4 11 6 7·6	55 5 61 0 48 9	55 5 71 2 47 6	—	—	9 lb 3 oz lb oz lb oz 13 3-5 12	9 lb lb oz. lb 16 10-6
38·9 49 0-32·5	37 3 42 5 31 0	9 8 11 7 8·7	9 9 13·0-8 9	57·5 63 3 50 0	58 2 61 6 53 2	—	—	11 lb lb oz lb oz 13 14-8 10	9 lb 12 oz lb oz. lb oz. 11 9-8 2
40·4 43 0-35 0	41·3 45 5 36·0	10·8 14·1-9·2	9·8 10·8 8 3	59·2 62·9-52·0	56 9 60 3-52 0	—	—	12 lb lb oz lb oz 15 4-9 8	11 lb 12 oz lb. oz. lb oz 13 7-9 12
41 6 48 1-36 8	41 5 45 5-35 9	10·7 12 2-9·8	10·7 12·0-9·5	61·6 65·5-58·4	63 8 71·1-61 6	—	—	13 lb 9 oz lb oz lb oz 15 8-12 10	13 lb. 9 oz lb oz lb oz 15 10-12 13
43 4 47 5-35·0	41 9 45 5-37 4	11·0 12·1-9·9	11·5 13 5-9·8	61·9 66·0-58 4	62 2 66 6-58 4	—	—	14 lb 9 oz lb oz lb oz. 16 3-12 3	15 lb 8 oz. lb oz lb oz. 17 9 12 3
44 0 50 0 37 0	46 6 56 5 38 0	11·4 13·1-9 5	12·9 14 4-10·6	65·0 69 2-51 5	66 2 73 0 62 5	—	—	16 lb 3 oz lb oz lb oz 20 11 14 6	16 lb 3 oz lb oz lb oz 21 0-14 12
47 6 53 0-36·5	47 0 54 5 39·0	12·4 13·3-10·0	12·9 18·2-10·7	68 1 72 4 61 0	70 1 76 8 62 9	—	—	20 lb 0 oz lb oz. lb oz 23 7-16 8	20 lb lb oz lb oz. 22 2-15 9
44 6 48 5 40 3	42 5 45 5-41·5	12·9 14·8-11 3	11·9 12·8-11 0	73 6 83 8 68 6	69 5 71 1 65 5	45·1 49·0-42·2	—	21 lb lb. oz lb oz 23 14-17 0	19 lb lb. oz lb oz 20 0-17 2
47 0 50 0-40 5	44 8 50 5 38 5	13·2 14 6-10 4	13·4 18·3-10·9	77·0 81 3-72 4	78·7 102 9-68 6	47·6 50·2-45·1	47·8 54·7-43·2	22 lb lb oz lb oz 27 14-19 7	21 lb 12 oz lb oz lb oz 34 0-16 0
48 1 56 5 43 2	45 7 52 0-41 5	13·9 17·2-12·0	13·2 16·2-11·3	80·4 97·8-73·7	76·6 85·1-66 1	47·9 50·8-45·7	47·1 51·5-44 4	23 lb. 8 oz lb. oz. lb oz 28 15-19 11	22 lb 8 oz lb. oz lb oz 25 11-19 10
46 9 54 2-39 5	47 8 53 5-43 6	14 0 22 5-11 1	14·2 15 7-13·1	80·2 88 8-69·8	88·6 92·0-73 6	49·9 53 2-47 0	49·2 54 6-44·5	25 lb lb oz. lb oz 30 7-20 7	24 lb lb oz. lb. oz 28 7-21 0
48 7 54 3-42 5	47 6 55 5 41 0	14·5 17 6-13·2	14·3 17 0-12 3	84·2 91 4-76 2	82·5 94·7-64·0	50·2 54·6-45·0	49·0 55·9-44 0	28 lb lb oz. lb oz 31 8-21 4	26 lb lb oz lb oz 29 10-21 0
48 2 53 5 40 1	48 4 54 0-42 0	15 1 16·9-14 0	14·5 18 4-13·3	88·7 95 0-78 7	87·4 91 4-80·7	51 8 55·9-46·3	50·6 59 8-46·3	28 lb. 8 oz lb oz lb oz 31 10-24 10	27 lb 8 oz lb oz lb oz 36 1-21 8
51 7 56 0-47 0	49·6 53 5 39 0	16·1 18 1-14·7	15 8 18·0-13·7	94·8 102·2-87 5	92·3 102 9-82 6	55·1 61 6-50 1	54·4 58 6-49 5	32 lb lb oz. lb oz 37 10-27 2	31 lb lb oz lb oz 38 0-25 6
50 5 60 3 44 5	50 5 58 5-45 5	16·5 19 7-14 8	16·5 19·0-14·9	96·5 107·9-77 2	99 5 107·9-89 5	56·7 62·2-47 0	56 7 67·7-53·2	32 lb 1 oz lb oz lb oz 41 2-26 6	33 lb lb oz lb oz. 40 9-27 13
51 5 56 5-46 0	50 7 57 0-44 2	17·6 24 0-15 6	17·2 19·8-15 9	106·0 120 6-92 7	105·2 119·3-95 9	60·1 68 6 52·7	59·6 66 0-54 6	36 lb 8 oz lb oz. lb oz 45 0-29 0	37 lb lb. oz lb oz 47 8-29 8
51 7 57 5-44 2	51·9 62 0-46 0	17 9 19 1 14·9	17·9 21·3-16·3	110·9 121·9-85·0	112·6 133·5-101·6	62·5 68·2-52 7	61·9 71·8-47·0	40½ lb lb oz lb oz 56 0-33 8	41 lb lb oz lb oz 57 2-31 0
53 0 56 5-46 0	52 4 59 4-45 0	18·9 21 1-16·3	18·5 21·0-16·4	117·2 126 3-104 6	117 5 135·5-102·8	65·2 69·2-59·1	65 2 74 3-57·7	46¼ lb lb oz. lb oz 55 0-34 8	44 lb lb oz lb oz 57 0-30 0
56·3 61·5-49 0	53 0 66 0-46 5	19 6 22 4 16 2	19·1 22 6-15 3	122 1 131 5-111 1	120·4 130 2-107 9	67·1 73 0-54 6	66·2 74 9-60 3	50 lb lb lb 60½-39	46½ lb lb lb 56½-33
54 5 61 0-49 7	53 6 62 3-49 0	20·0 21 9-18 0	19·6 24 6-18·4	125 4 140·3-113·6	125·2 142·9-113·0	69 1 76·9-62 2	68·4 74 3-60 3	53 lb lb. lb. 71-41	50½ lb lb. lb. 65-38
58 3 64 5-54 0	55 0 68·0-48·2	19·9 21 9 18 1	20·5 26 7-16 9	129 5 135 9 124 5	130 0 140 9-121 2	72 4 94 7-66 6	70 0 75 5-64·0	56 lb lb lb 67-38	56 lb lb lb 73½-41
56 4 62 5 49 0	55 8 62 9-51 0	20·8 22·7-18 8	20·6 23 2 18 0	133 0 140 9-126 3	135 0 146 6-114 9	72 6 92·7-53 9	72 0 78 1 51 5	62 lb 1 oz lb lb 73¾ 48½	64 lb 3 oz lb. lb 76-43
60 3 74 5-48 0	59·1 65 5-53·0	21·4 24·8-18 7	21 5 25 6-15 5	140 3 166 2-116 2	139 1 156 9-119 3	76 3 99·7-64 1	75 7 94 0-69 2	73 lb lb lb 115 61	68½ lb lb lb 86-54
61 3 67·3-55 5	58 7 68 3-49 5	21·5 23·1-20·0	21·9 25 7-16 6	138 3 149·2-124·2	147·1 160·6-140·3	74·2 78·7-67·5	76 1 81 3 71 7	72 lb lb lb 85-61	74½ lb lb. lb 82-53

for each sex; the figures beneath the averages indicate the extremes obtained.

the effects of obstetrical moulding have passed off, show that although the index slightly decreases with increasing age owing to the growth of the frontal sinuses, yet the type of the head remains the same—a dolichocephalic one remains dolichocephalic, and a brachycephalic one remains brachycephalic throughout life. Thus, in Table CCXIIIA the index is mainly of the dolichocephalic type from birth onwards, because the English are a dolichocephalic race.

(iv) *The Capacity of the Cranium.*—Berry[1] gives the following table stating the cranial capacity at different ages.

TABLE CCXIV.

Age	Capacity of cranium in cubic centimetres		Percentage volume of brain		Phase of sexual life
	Boys	Girls	Boys	Girls	
Birth ..	371	333	25	25	Pre-pubescent phase
1 year ..	945	849	63·7	63 7	
2 years ..	1,075	966	72·5	72·5	
3 ,, ..	1,151	1,035	77·6	77·6	
4 ,, ..	1,186	1,066	80·0	80·0	
5 ,, ..	1,206	1,096	81·3	82·2	
6 ,, ..	1,225	1,121	82·6	84·1	
8 ,, ..	1,264	1,162	85·2	87·1	
10 ,, ..	1,301	1,199	87·7	89·9	
12 ,, ..	1,326	1,226	89·4	91·9	Resting phase
14 ,, ..	1,358	1,271	91·6	95·3	Pubescent phase
15 ,, ..	1,378	1,279	92·9	95·9	Post-pubescent phase
17 ,, ..	1,422	1,305	95·9	97·9	
20-30 years	1,483	1,333	100·0	100 0	

The cranial capacity can be estimated in the living person from a knowledge of the length, breadth and height of the head.

(*e*) **Thoracic Circumference.**

(i) *The average thoracic circumference at birth* is about 34 cm. (13·6 in.).

(ii) The circumference (*α*) increases by about 50 per cent. at 2½ years ; (*β*) doubles itself at 13 to 14 years.

(*f*) **Abdominal Circumference.**

(i) *The average abdominal circumference at birth* is about 33·5 cm. (13·4 in.).

[1] R. J. A. Berry, "The Stewart Lectures," University of Melbourne, 1925.

(ii) The circumference (α) increases about 50 per cent. at $2\frac{1}{2}$ years; (β) increases by about 90 per cent. at 13 to 14 years.

(*g*) **Vital Capacity** (i.e., the maximum amount of air it is possible to take into the lungs in one breath as measured with a spirometer). This, of course, is impossible to ascertain in infants, but several people have measured it in children from 4 years upwards.

Stewart[1] gives the following table :—

TABLE CCXV.

Age in years	Boys			Girls		
	No. of cases	Standing height in cm.	Vital capacity in c.c.	No. of cases	Standing height in cm.	Vital capacity in c.c.
4	6	103·4	792	9	95·4	664
5	20	106·8	927	26	106·4	888
6	62	112·2	1,154	62	111·5	1,085
8	98	121·8	1,468	76	121·0	1,401
10	87	133·4	1,872	117	132·1	1,672
12	114	142·4	2,182	135	144·0	2,053
14	177	154·8	2,712	192	156·6	2,607
16	67	167·2	3,425	29	160·1	2,778
18	9	172·0	3,922	1	162·0	3,100

Dreyer gives the following formulæ expressing the relationship between vital capacity, and other anthropometric measurements in the two sexes at any age.

$$\text{Vital capacity} = \frac{W^{0\cdot72}}{0\cdot69} \text{ in boys, or } \frac{W^{0\cdot72}}{0\cdot79348} \text{ in girls (W = weight in grm.)}$$

$$= \frac{\lambda^{2\cdot257}}{6\cdot1172} \quad ,, \quad \text{or } \frac{\lambda^{2\cdot3003}}{8\cdot2714} \quad ,, \quad (\lambda = \text{sitting height in cm.})$$

$$= \frac{Ch^{\cdot1973}}{1\cdot5595} \quad ,, \quad \text{or } \frac{Ch^{2\cdot5352}}{16\cdot4951} \quad ,, \quad (Ch = \text{thoracic circumference in cm.})$$

Rates of Growth at Different Ages.—C. H. Stratz[2] finds that growth in height is most rapid during the 1st year, falls slightly during the 2nd year and rises again at the end of the 4th year. At the end of the 6th year, as well as at the 11th year, there is a particularly marked acceleration (see fig. 149). When growth is most rapid, the child's energies in other directions must be restricted and his food must be more abundant. His clothing also must be adapted to the extra growth.

[1] *Amer. Jour. Dis. Ch.*, June, 1923.
[2] "Der Körper des Kindes," Stuttgart, 1921.

Characters of Growth in the Two Sexes (fig. 149). (1) Up to the end of the 11th year the two curves for males and females run practically parallel courses, that for females being slightly

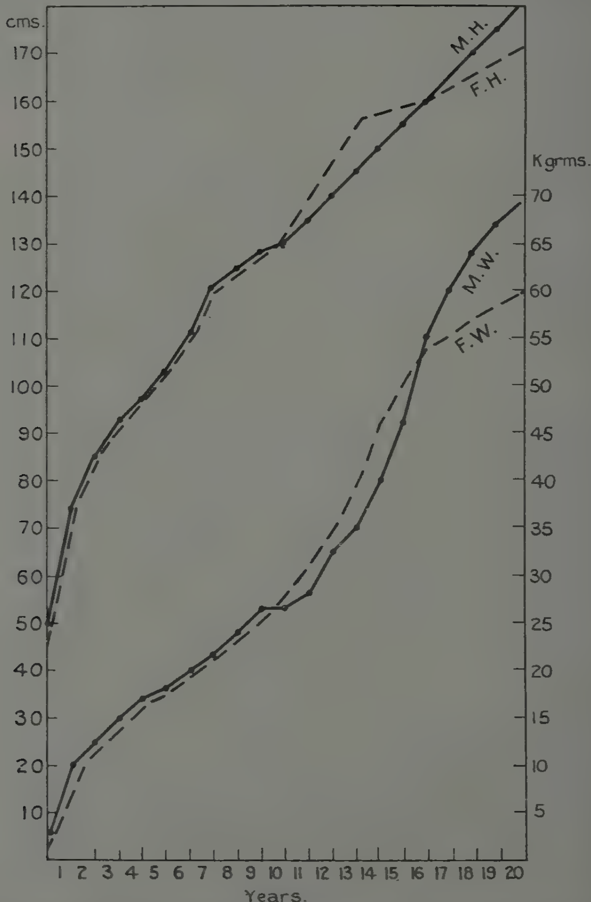

FIG. 149.—Growth curves from birth onwards. F. = female; M. = male; H. = height; W. = weight.

lower than that for males. (2) The rise at the end of the 11th year is higher in females than in males, so that girls from that age till about the end of the 16th year are actually taller

than boys of the same age. The curve for girls crosses that for boys and lies on a higher level than the latter during that age period. At sixteen years the two curves recross and the curve for females again assumes a lower level.

The weight curves are essentially similar to those of height.

X-ray examinations have shown that ossification of bones occurs earlier in girls than in boys (by about 6 months at 5 years and by about 2½ years at 15 years).

The following table represents the annual increase in height and weight from birth onwards as given by Stratz :—

TABLE CCXVI.

Year	Annual increment in Height (cm.)		Annual increment in Weight (kgm.)		Increase in kgm. per cm.	
1	25		6		0·25	
2	10		3·5		0·35	
3	8		1·5		0·20	
4	4		2		0·50	
5	6		1·5		0·25	
6	8		1·5		0·20	
7	10		3		0·30	
8	4		2		0·50	
9	3		2		0·75	
			M.	F.	M.	F.
10	2		0·5	1·5	0·25	0.75
	M.	F.				
11	5	8	4	5	0 80	0·63
12	5	5	2·5	3	0 50	0·60
13	·6	12	4	4·5	0·65	0.40
14	5	3	3	6	0 60	2·00
15	9	2	7	6	0·75	3 00
16	2	2	8	1	4·00	0·50
17	3	1	5	2	1·60	2.0
18	5	2	4	1	0 80	0 50
19	5	3	3	1	0·60	0·30
20	5	2	2	0·5	0·40	0·25

B. BODILY PROPORTIONS. (See fig. 150.)

(*a*) **Relation between Cranial and Thoracic Circumferences.**

The following table expresses the change in this relationship with growth (the numbers represent centimetres) :—

TABLE CCXVII.

Age	Head circumference / Thorax circumference
Birth	36 : 34
6 months	42 : 42
2 years	49 : 50
13-14 years	54 : 67
Adult	55 : 68

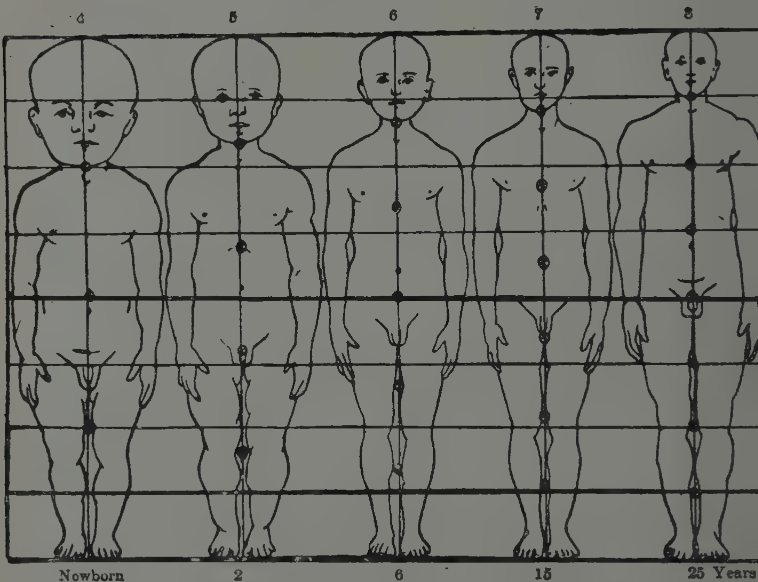

FIG. 150.—Proportional growth from infancy to twenty-fifth year (Stratz).

We see, therefore, that at birth the cranial circumference slightly exceeds the thoracic; between 6 months and 2 years the two measurements are about equal; whilst after that the thorax grows much more rapidly than the head. It has been said that amongst infants, in whom the cranial measurement at birth is not larger than the thoracic, there is a higher stillbirth-rate as well as a higher neo-natal mortality-rate (see p. 502). Also, if after the 2nd year the thoracic circumference does not exceed

the cranial, there is something wrong with the child's normal development: it is probably ricketty or possibly even hydrocephalic.

(*b*) **Relation between Sitting Height and Standing Height.**—The following table represents the approximate percentage that the sitting height forms of the total height at various ages:—

TABLE CCXVIII.

Age	Proportion per cent. of sitting height to standing height	Percentage ratio length of lower limbs and standing height
Birth	68 per cent.	32
6 months	63 ,,	37
1 year	63 ,,	—
2 years	60 ,,	40
13-14	54 ,,	46

From this we see that at birth the trunk is much longer than the lower limbs, whilst as the child grows the length of the lower limbs which, at birth, forms only 32 per cent. of the total length of the body, increases more rapidly than the trunk until in adult life the legs attain a length of about half that of the total height.

Many cases of dwarfism are due to failure of the legs to grow at their normal rate.

Faulty environment retards the growth of the lower limbs and makes the ratio between sitting height and standing height greater in children of a lower social level than in others.

(*c*) **Relation between Sitting Height and Weight of Body.** Dreyer finds that at any age the relation between weight in grammes (W) and the sitting height in cm. (λ) is given by the formula:—

$$W = (0{\cdot}38025\,\lambda)^{\frac{1}{0{\cdot}319}} \text{ in males}$$

$$= (0{\cdot}662\,\lambda)^{\frac{1}{0.365}} \text{ in females}$$

(*d*) **Relation between Thoracic and Abdominal Circumferences.** At birth, and up to the age of about 3 or 4 years, the abdominal circumference is approximately equal to that of the thorax. After that age the abdomen loses fat, and its circumference gradually becomes less than that of the thorax.

Stratz professes to be able to tell the age of an individual from his photograph, by measuring the proportions of the various parts of his body.

C. Growth in Different Organs.

So far we have been speaking of the growth of the body as a whole, but the various internal organs or viscera of the body vary considerably in their rates of growth. All organs with the exception of the suprarenals and the thymus (which gradually diminish in absolute size and weight as the body grows) increase in absolute weight as the child passes from infancy to puberty. Their rates of growth, however, are not the same. Thus the muscles grow twice as quickly as the body as a whole; the skeleton, the lungs, the gastro-intestinal tract and spleen retain their original proportionate weights to the body as a whole, while others, like the liver, the heart, the kidneys, &c., lag behind the general body growth. Table CCXIX shows the rate at which each of these organs grows. From this it is seen that the brain is the organ whose rate of growth is least, so that instead of being one-seventh of the body weight at birth it is no more than one-fiftieth of the body weight in the adult. During the period of adolescence, the heart, the muscles and the sex organs grow with increased activity. The heart, for instance, doubles in volume at that time, as seen from the following figures representing the volume of the heart at different ages: At birth 20 c.c., at 1 year 40 c.c., at 3 years 57 c.c., at 7 years 87 c.c., at 13 years 120 c.c., and in the adult it is more than 260 c.c. The growth of the heart volume during adolescence from 120 c.c. to 260 c.c. is important, because it keeps pace with the increase of muscular activity during that period, and thus minimises any risk of cardiac overstrain.

The following table shows the percentage weights of the various organs (in proportion to the body as a whole) in the infant and adult.

Table CCXIX.

Organ or system	Newborn infant	Adult
Skeleton	16·7 per cent.	15·35 per cent
Muscles	23·4 ,,	43·09 ,,
Skin	11·3 ,,	6·30 ,,
Brain	14·34 ,,	2·37 ,,
Spinal cord	0·20 ,,	0·07 ,,
Eyes	0·28 ,,	0·02 ,,
Heart	0·89 ,,	0·50 ,,
Thymus	0·54 ,,	0·01 ,,
Liver	4·39 ,,	2·80 ,,
Suprarenals	0·31 ,,	0·01 ,,
Testes	0·04 ,,	0·08 ,,

When, therefore, Longfellow called the child a man's counterpart in miniature he was incorrect. Rather, as Bishop Earle, in the quotation given at the head of this chapter, has put it: "The child is a man in a small letter" (as compared with the adult who is a man in a capital letter). Cicero probably recognized this long ago when he wrote: "Nature conducts us by a regular and sensible progression through the different seasons of human life, to each of which she has annexed its proper and distinguishing characteristics."

Factors which influence Physical Growth.

(1) **Race and Heredity**. Some races, like the Patagonians, the North American Indians, the Scotch, the Swedes and Norwegians, are particularly tall, whilst others, like the Japanese, the Esquimaux, the South Italians and Spaniards, are short. It is not quite certain, however, as to how far the height in these races is determined by the differences in their environments, especially in regard to their food. Holt, for instance, states that Japanese children, born and bred in the United States, grow up taller and heavier than the native Japanese of the same age, and believes this to be due to the greater amounts of protein taken by the former. Similarly, the Australian-born children of English parents are taller and heavier than native English children (Robertson). On the other hand, until we know the physique of the parents of these American-born Japanese or Australian-born English children it would be rash to jump to any conclusion. It is quite conceivable that these parents may be of better physique than the native Japanese or English (because it is the physically stronger that generally venture abroad) and that the better physique of their children may be truly a racial rather than an environmental character. That environment, however, is at any rate partly responsible for it is shown by the fact that the American-born children of Jewish immigrants are, according to Fishberg, 3·7 cm. taller than their parents (i.e., have an average height of 167·9 cm., as against that of 164·2 cm. of their parents[1]). The dietetic experiments of McCarrison (see p. 353) offer an explanation of the exceptionally fine physique of certain of the Indian tribes.

As an example of the influence of heredity, Stanley Hall quotes Wiener, who recorded the measurements of his four sons. He found that in the case of three of them who had the same

[1] See Feldman, W. M., "The Jewish Child," 1917, p. 414.

mother, the height-age curves were very much alike, but in the case of the fourth who had a different mother, the curve was of a different character. Indeed, Pearson gives the correlation coefficient for height between parent and child as 0·505. But this high correlationship only applies to adults; in the case of infants, Miss Clark,[1] who calculated it for children between 2 weeks and 14 years, the coefficient is negligibly small —the highest one being 0·0758 ± 0·025 (between the height of the daughter and that of her mother). In other words, during the period of growth, environment appears to have a greater influence upon height than has heredity.

The following table shows the influence of race upon birth-length :—

TABLE CCXX.

Parentage of child	Birth length in cm.	
	Male	Female
American	52·5	52·3
English	49·6	49·1
German	51	49
French	49·9	49·2
Japanese	49	48 (2)

But even here it is not quite easy to exclude the influence of the maternal diet.

(2) **Sex.**—Female children are at all ages shorter and lighter than the male. The accelerated growth in males may possibly be due to the greater cell-division occurring in their sexual glands which determines a greater flow of blood to the neighbourhood, and this increased circulation accelerates growth not only locally, but also throughout the body. It has been calculated that during a lifetime, man forms 340 billion spermatozoa as against no more than 100,000 ova.

(3) **Environment.**

(*a*) *External Environmental Factors.*

(i) *Food.*—Russov in 1879 was the first to point out that breast-fed infants grew more rapidly than those fed artificially. Numerous other observers have since then come to the same conclusion, but recent statistical

[1] Med. Res. Council Special Report Series, No. 101, 1926.

[2] Y. Yoshido, "On the Development of Body Length, &c., of the Japanese." *Japan Medical World*, vi, 1926, p. 270.

research has shown that the prejudicial influence of bottle feeding does not extend beyond the 10th month of life. Thus whilst the correlation coefficient between weight and duration of breast feeding in the case of Glasgow infants is $0·3151 \pm 0·043$ at the age of 4 to 5 months, it is $0·2628 \pm 0·047$ at 9 to 12 months and only $0·0191 \pm ·0065$ at 1 year. It is probable that the diminution in the size of the coefficient is due to the gradual elimination by death of the less fit amongst the bottle-fed babies, with the result that after the end of the first year the comparison is made between a random sample of breast-fed babies and a selected sample (viz., the healthiest) of non-breast fed. As regards increase in length of infants, there is no marked difference between breast-fed ones and those fed on the bottle. Indeed it has been shown that animals as well as infants can grow in their linear dimensions, although the amount of food they receive is not sufficient to maintain their weight—the energy for that inherent growth impulse being derived from the combustion of the body fat.

Milk and Body Growth. We saw on p. 402 that the amounts of proteins and salts in the milks of various animals are in proportion to the rates of growth of the young of these animals. Yet the growth of a baby fed on cow's milk is not accelerated to that of the calf. In other words, it is not the milk which determines the rate of growth of the animal, but the rate of growth of the young which has determined the nature of the milk in the mother animal.

The relationship between the various foodstuffs (including vitamins and amino-acids) and growth has been sufficiently dealt with (see pp. 363 *et seq.*). The extra amount of food required for growth at various ages has been discussed on pp. 356-362. Here it will be of some interest to consider the **relationship between the amount of food taken by a child throughout its growth period and the rate of its growth** :—

On *a priori* grounds one might expect that the more calories a child can take per day, the more rapid would be its rate of growth. This is not found to be the case. Thus, *when the calorie value of the food is above a certain minimum* (i.e., above 2,500 per adult man and *pro rata* for women and children), the partial correlation coefficient (see pp. 77-79) between weight (1) and calories (2) for constant age (3) is either insignificant (as in the case of Derbyshire miners) or small as in the case of Glasgow.

Glasgow	.. .. ..	Boys	$r_{12\cdot3} = \cdot3085 \pm \cdot062$
		Girls	$r_{12\cdot3} = \cdot2700 \pm \cdot058$
Miners (Derbyshire)	..	Boys	$r_{12\cdot3} = \cdot0844 \pm \cdot081$
		Girls	$r_{12\cdot3} = \cdot0124 \pm \cdot081$

The explanation for this practical independence between amount of food and weight (when the number of calories is beyond a minimum) lies in the fact that when the amount of food is increased beyond a certain optimum it becomes incapable of being digested, absorbed and metabolised.

The Minister of Health recently made a statement in the House of Commons that when school children were given a pint of milk in addition to their ordinary food they grew 50 per cent. more in height, and 100 per cent. more in weight than those who have no such addition during the same period of time. It is difficult to reconcile such a statement with the statistical and experimental findings discussed above, except in the case of children who did not receive the minimum number of calories at home, and the subject needs very careful investigation conducted on a large scale and in a scientific and unbiased manner.

(ii) *Social Position.*—Numerous investigations have shown that the children of the poorer classes have, age for age, a lower weight as well as a lower height than those of the rich, and at first sight it would appear that this is entirely due to the difference in the economic environments of the two classes. More penetrating statistical research has shown, however, that such an explanation is not altogether adequate, and that the hereditary element is probably responsible for a considerable share of this difference. For when the effect of heredity is eliminated by comparing children belonging to the same social states (say the slums) but differing only in respect of the size of their parents' incomes, the differences in their heights and weights become considerably less noticeable. Indeed even without eliminating the hereditary factor, figures show that although the richer children are, age for age, better and heavier than those of the poor, yet per unit of height the rich children are lighter than the poor.

The environmental condition associated with slum life is poverty, with its concomitants of underfeeding, &c., as well as overcrowding and insufficient air space When the influence of each of these factors is analysed,

it is found that, *when a certain minimum has been surpassed*, there is no appreciable difference between children who are better off in respect of each of these conditions than those who are less favourably situated.

(*a*) *Poverty.*—Annabel M. Tully,[1] has shown that neither during the age-period 0 to 5, nor during the period 6 to 14, is the parents' income (beyond a certain minimum) in the Glasgow, Edinburgh or Dundee slums considerably correlated with either the height or the weight of their children. In other words, her results do not indicate that a mere increase of wages would, *per se*, lead to a better growth of the children.

(*β*) *Underfeeding.*—We have already seen (p. 639) that when a certain minimum number of calories is exceeded, the relationship between calories and either heights or weights of children is very slight. Miss Tully and Miss E. M. Urie[2] have shown that when the family income rises above the poverty line, i.e., the line below which the necessities of life *alone* can be procured, the diet, although it frequently becomes *actually* more liberal, nevertheless contains *proportionately* less calories per unit of expenditure because more expensive foods are indulged in, as seen in Table CCXXI.

TABLE CCXXI.

Income per man per week (1)	Expenditure on food per man per week (2)	Calories per penny (3)	Total calories (4)
12s.	9s. 1¾d.	173·8	2446
6s. 10½d.	6s. 1¼d.	219·8	2505
5s.	3s. 10½d.	302·3	2182

P. L. MacKinley[3] finds that the coefficient of correlation between total income per man and the calories purchased *per penny* is negative and

[1] "The Physique of Glasgow School Children." *Journ. Hyg.*, xxiii, 1924, p. 186.

[2] "A Study of the Diets and Economic Conditions of Labouring Class Families." *Glasgow Med. Jour.*, xvi, 1922, p. 353.

[3] "Anthropometric Studies of Glasgow School Children." *Journ. Hyg.*, 1924, xxiii, p. 176.

large, viz., $r_{13} = -\ \dot{}5982 \pm \dot{}063$, although the coefficient between the total income and the *total* calories purchased is positive and also large, viz., $r_{14} = \dot{}5474 \pm \dot{}069$. The correlation coefficient between weight of child and calories per penny is in most cases insignificant (e.g., in the case of Derbyshire miners' girls = − ·1003 ± ·080) except in the case of Glasgow and agricultural boys in whom it is − ·4218 ± ·057. This means that, in most cases, the goodness of marketing has little, if any, relation to the weight of children, possibly because the extra number of calories purchased is discounted by the worse quality of the food in respect of amino-acids and vitamins (e.g., vegetable proteins and fats). Thus, although there is no significant correlationship between the amount of protein and the number of calories either per unit of income (− ·1117 ± ·111) or per unit of food expenditure (·086 ± ·146), yet the proportion of vegetable protein (as well as of vegetable fat) is greater in the dietaries of people with lower incomes—as seen from the following Table. (The figures in brackets represent the percentage amounts of proteins and fats from vegetable sources.)

TABLE CCXXII.

Income per man per week	Expenditure on food per man per week	Calories per penny	Diet per day			
			Protein grm.	Fat grm.	Carbo-hydrate grm.	Calories
21s. 8d.	12s. 5d.	159	96 9 (48·71)	128·9 (16·93)	459·3	3479·5
10s.	6s. 5d.	279	78·8 (65·23)	99·6 (44·85)	456·1	3119·1
6s. 2½d.	4s.	278	44·0 (76·29)	42·3 (56·68)	332·2	1939·9

Hence, although the purchasable number of calories per penny rises with fall of income, the growth value of the food per penny is less, with the result that the nutrition of the child is within certain limits independent of the income.

The following are other interesting coefficients :—

Income/carbohydrate	=	$-\cdot6203 \pm \cdot669$
Income/fat	=	$\cdot4108 \pm \cdot093$
Calories per penny/carbohydrate ..	=	$\cdot5021 \pm \cdot110$
Calories per penny/fat	=	$-\cdot5532 \pm \cdot102$

In non-mathematical language these coefficients mean that as the income rises the proportion of the more expensive fat rises and that of the less expensive carbohydrate falls, and the opposite is the case with the calories per penny ; as they rise (with fall of income) the proportion of the cheaper carbohydrates rises and that of the dearer fats falls.

(γ) *Overcrowding and Insufficient Air Space.*—On *a priori* grounds one would expect that the domestic overcrowding associated with poverty, would, on account of the concomitant decreased air supply per person, have a prejudicial effect upon growth. This again is not entirely borne out by facts. Thus during the first year of life, during the pre-school as well as the school period, the correlations between domestic air space and the heights or weights of children, though in some cases significant, are, on the whole, very small. The explanation of this is that the house does not determine the total amount of fresh air breathed by the members of the family, since a considerable time is spent out of doors.

It is, however, necessary to bear in mind the possibility that in view of the high correlation between overcrowding and infant mortality (see p. 166), the absence of correlation between overcrowding and growth may be due to the survival of the fittest.

(δ) *Maternal Efficiency, or Good Mothering*—The following coefficients show that although the correlation between maternal efficiency and the child's nutrition is not very large, yet it is undoubtedly significant ; *e.g.*, in the case of Edinburgh children, 6 to 14 years old, the coefficients are :—

Boys :	Weight/maternal efficiency	$\cdot2317 \pm \cdot049$
	Height/maternal efficiency	$\cdot2763 \pm \cdot047$
Girls :	Weight/maternal efficiency	$\cdot2944 \pm \cdot044$
	Height/maternal efficiency	$\cdot1807 \pm \cdot047$

> Indeed maternal efficiency seems to be the dominant factor in relation to the nutrition of the child. Maternal efficiency seems to be uncorrelated with income, but is definitely correlated with the amount of air space per person in the house. There is no appreciable correlation between maternal efficiency and the number of living children in the family.

(iii) *Maternal Health.*—Closely associated with the question of maternal efficiency is that of maternal health, which "shows a small correlation with the weight and height of the children at different ages, but when the factor 'maternal efficiency' is kept constant this disappears. Maternal health is therefore not revealed as a factor of prime importance."[1]

(iv) *Seasonal Variations.*—During the summer months children grow more than during the winter—both in height and in weight—especially in height. Daffner, for instance, found in the case of cadets, aged 11 to 20, that the increases during these seasons were in the proportion of 3 : 2, especially among the younger boys. Holt found a greater increase in both weight and height of children during the summer, and Hildegarde Frank[2] found the same to be the case with babies. That this phenomenon is not entirely due to the greater opportunities for an open-air life in the summer is shown by the fact that the children at open-air schools are not better off in respect of growth than those in ordinary schools. It is probably in some way associated with the higher atmospheric temperature which increases the chemical reactions of the body resulting in growth. The growth of the tadpole, for instance, can be controlled at will by altering the temperature of the medium in which it lives.[3] Another factor is the greater abundance of ultra-violet rays, as shown by Miss Hume,[4] who fed two series of rats on a diet free from vitamin A and exposed one series to the rays of

[1] Med. Res. Council, Special Report Series, ci, 1926, p. 303.
[2] *Arch. f. Kinderheilk.*, 1924.
[3] See W. M. Feldman, "Child Physiology," p. 43.
[4] *Lancet*, December 25, 1922, p. 1318.

a mercury quartz vapour lamp, when she found that the rate of growth in them was greater than in the rats not so irradiated. The observations on Smethwick children (ch. xx, p. 486) are also of interest in this respect—although they need confirmation.

(v) *Illness*, if temporary, retards growth for the time being, but during convalescence there is generally a great acceleration in the rate of growth sufficient to make up for the previous retardation. In young babies, vaccination, dentition, and diarrhœa may cause a temporary loss of weight. Permanent illness such as heart disease necessarily causes a permanent retardation in growth.

(vi) *Diseases of nutrition, e.g.*, rickets, the tuberculous diathesis, &c.

(vii) *Physical defects*, especially naso-pharyngeal obstruction (adenoids) retard growth by preventing proper oxygenation of blood and restful sleep, until these defects are remedied.

(*b*) *Internal Environmental Factors.*—The internal secretory or endocrine glands have a profound influence on growth.

(1) *Pituitary.*—This consists of two lobes, anterior and posterior, each of which has its own internal secretion.

(α) If there is excess of secretion from the anterior lobe commencing in childhood before the ossification of the epiphyses, there is skeletal overgrowth resulting in gigantism. If the excessive secretion occurs in middle life after ossification is complete, then there results the condition called acromegaly.

(β) If there is defect of secretion from the anterior lobe, there is an arrest of skeletal growth, giving rise to the Lorraine type of infantilism (small body, small fine bones, loss of strength, &c.).

(γ) If there is excess of secretion from the posterior lobe, there is alertness of intelligence and possibly some glycosuria.

(δ) If there is defect in secretion from the posterior lobe, there results the condition known as Fröhlich's syndrome of pituitary infantilism, or dystrophia adiposogenitalis.

(2) *Thyroid.*—If there is deficient secretion occurring during the period of growth, there results the condition known as cretinism, viz., a stunting of physical and mental growth. Excessive thyroid secretion is exceedingly rare in childhood, except in girls at about the age of puberty (13 to 17 years).

(3) Infantilism may also occur as the result of *pancreatic* deficiency, or kidney disease occurring in early childhood, as well as in cœliac disease.

(4) *Suprarenals.*—Hypertrophy of the cortical layer in children is associated with excessive growth of the body and precocious sexual development. Atrophy of the cortical layer results in a kind of infantilism which Hastings Gilford has called *progeria* (the Greek for premature senility).

For the influence of other internal secretions upon growth, see "Child Physiology," Chapter XXXVIII.

Assessment of Nutrition.—Before we can attempt to estimate the state of nutrition of any given child we must have a clear idea of what is meant by the term. Unfortunately the physiologist and the physician are not in entire agreement upon the matter. The former basing himself on the fact that a deposit of fat in the body must necessarily imply good absorption and assimilation on the part of the child will call a fat child "well nourished." On the other hand the physician, paying special attention to the poise of the body, the tone of the muscular and nervous systems as shown by the child's easy fatiguability, irritability of temper and capriciousness of appetite, his colour (whether pale or otherwise), disturbance of sleep, &c., would quite possibly classify the same child as undernourished. Hence we see that "weight for age" is not in itself a satisfactory *index of nutrition*. Similar remarks apply to "height for age," because not only is height to a considerable extent a racial and hereditary character, so that what should constitute the *normal* height for any particular child may be above or below the average height as found from the tables (which are based upon the examination of many children of different classes and nationalities)—but we know that growth in height continues even when the total food energy supplied to the child is insufficient, because the child grows in height at the expense of its own tissues. Hence the child may be tall and undernourished.

In order to supply the family doctor, the School Medical Officer, and the educationist with a method of identifying the

state of nutrition of a child which would eliminate any possibility of the same child being placed into different nutritional categories varying with the training or the personal equation of the observer, different people have from the time of Quetelet onward expressed the index of nutrition by different formulæ made up of functions of two anthropological measurements. So far, however, no formula yet proposed has proved infallible.

The following are a few indices of nutrition suggested :—

$\frac{100\ W}{H}$ (Quetelet). This gives the weight, per cent. of the height.

$\frac{100 \sqrt[3]{W}}{H}$ (Livi, 1899). This formula was put forward on the idea that weight being proportional to H^3 it is unreasonable to compare a tridimensional with a unidimensional unit.

$\frac{100\ W}{H^3}$ (Buffon, 1833, and Rohrer, 1908). This formula is based upon similar considerations.

$\frac{100 \sqrt[3]{10W}}{\lambda}$ (Pirquet, 1916). Pirquet finds that λ (i.e., sitting height) = $\sqrt{\text{intestinal area}}$, and that $\sqrt[3]{10W}$ is also equal to $\sqrt{\text{intestinal area}}$, so that $\frac{100 \sqrt[3]{10W}}{\lambda}$ should be equal to 100. This he finds to be the case at all ages after growth had ceased, but that during growth this ratio, which he calls *pelidisi* (*p*ondus decies *l*inearis *d*ivisa *s*edentis a*l*titudine) should be equal to 94. Thus, if $\lambda = 60$ cm. and $W = 12{,}500$ grm. then the index $= \frac{100 \sqrt{125{,}000}}{60} = 83{\cdot}3$, so that the child is undernourished.

Mackinley, using the data collected from the case sheets of the Royal Hospital for Sick Children, Glasgow, tried to correlate the senior physician's estimate of the child's nutrition with some of the indices suggested and he found that Quetelet's formula $\frac{100\ W}{H}$ is more closely related with the physician's idea of nutrition than any other. The following Table gives some of his results.

TABLE CCXXIII.—CORRELATION OF INDICES WITH NUTRITIONAL ESTIMATE (ALL AGES).

	Weight	Height	W/H	W/H³	W/A
Boys..	·4266 ± ·0225	·3956 ± ·0232	·6489 ± ·0159	·4993 ± ·0207	·6007 ± ·0179
Girls	·6105 ± ·0201	·3015 ± ·0291	·6469 ± ·0186	·4049 ± ·0268	·5809 ± ·0216

A weight of more than 10 per cent. under the average weight for the height is taken to be as evidence of malnutrition.

TABLE CCXXIV.—WEIGHT TO HEIGHT INDEX AT VARIOUS AGES IN THE CASE OF TORONTO CHILDREN. (AFTER ALAN BROWN.)

Age	Boys			Girls		
	Weight (lb.)	Height (inches)	100 W. / H	Weight (lb.)	Height (inches)	100 W / H.
Birth	7·55	20·6	36.7	7·16	20·5	34·9
3 months ..	11·8	23·5	50·2	11·3	23·2	48·7
6 ,, ...	16.0	26·5	60·4	15·5	26·0	59·6
1 year	20·0	28·8	69·4	19·5	28·2	69·5
2 years	26·5	32·8	80·8	26·0	32·2	80·7
3 ,, ...	31·0	35·7	84·0	30·5	35·2	83·8
4 ,, ...	35·0	38·2	91·1	34·3	37·7	91·0
5 ,, ...	39·0	40·6	96·0	38·0	40·2	94·5
6 ,, ...	43·0	42·8	100·0	41·6	42·3	98·3
7 ,, ...	47·4	45·0	105·3	45·5	44·5	102·3
8 ,, ...	52·0	47·4	109·7	50·1	46·7	107·3
9 ,, ...	57·0	49·4	115·4	55·0	48·7	113·0
10 ,, ...	62·5	51·0	122·6	60·2	50·7	118·7
11 ,, ...	68·0	52·7	129·0	66·1	52·7	125·4
12 ,, ...	73·7	54·5	135·4	74·1	54·9	135·0
13 ,, ...	80·8	56·5	143·0	83·7	57·3	146·1
14 ,, ...	90·0	58·8	153·0	93·5	59·5	157·1

Gray and Edmands[1] found that Pirquet's index gave an error of 21·5 per cent., and Faber points out that an error of 5 cm. in the sitting height—which is quite easily made in view of the dependence of this measurement upon the state of contraction or relaxation of the thigh muscles—may alter the value of the index by 8 to 10 points. The same remarks apply to Dreyer's indices (see pp. 631 and 635), and Rodgers[2] finds that the correlation between sitting height and vital capacity is not better than

[1] *Amer. Journ. Dis. Child.*, xxiii, 1922, 226.
[2] *Arch Int. Med.*, xxxi, 1923, 342.

between standing height and vital capacity. Murford and Young[1] came to the same conclusion. Hence we may say that the best index of nutrition is W/H, but that in addition it must be fortified by a physical examination of the child with respect to its muscular and nervous tone, the poise of the body, &c. Were we to rely upon this index alone we may occasionally arrive at the anomalous conclusion that although a child is both in respect of height, weight, and physical examination of relatively high standard of nutrition, yet it may not be quite so heavy for its height as would be required by the W/H ratio. Thus all investigations show that the children of the better social classes brought up under the most favourable surroundings are age for age taller and heavier than those of the lower social classes brought up under less favourable surroundings, yet they are relatively lighter for their height than the poorer children. This however may be due to pampering which prevents the well-to-do children from benefiting from their food as much as the poorer children. Indeed one can say fairly definitely that malnutrition in children is not as a rule due to lack of food but to the wrong kind of food. Another possible factor is the greater mental pressure and overstrain to which they are subjected at school, which leads to fatigue, lack of sufficient sleep and its consequent malnutrition. The importance of fatigue as a factor in malnutrition is shown by the fact that some undernourished school-children fail to gain weight even when supplied with extra nourishment—until they are given a rest after their extra meal.

Posture.—For the maintenance of the erect position of the human body the extensor muscles must be suitably developed, and as soon as any of these muscles fail, either as the result of fatigue, or of faulty nutrition, the body assumes a bent and slouching posture. The spine becomes curved, the chest becomes cramped and the aeration of the lungs is impeded. The abdominal organs tend to drop, giving rise to intestinal stasis with its consequences. Hence faulty posture is a cause of malnutrition.

In the correct posture, the child stands erect, with the shoulders back. The head is erect on the trunk and the chin is held back. The chest is thrust forward and the abdomen is flat. The legs are straight. In the incorrect posture the head slants forward with the chin dropped, the shoulders are rounded and

[1] "Biometrika," xv.

may be of unequal level: the chest is flat and the abdomen protruding, the legs are bent (fig. 151).

Incorrect posture may be of congenital origin but is in the vast majority of cases due to faulty environments, rickets, infantile paralysis, adenoids, faulty vision, lack of exercise, especially of the trunk muscles, and too heavy clothes or improper footwear. It is not easy to say whether bad posture is a result of fatigue, or fatigue is the result of faulty posture, but that the two are almost constantly associated together is quite certain.

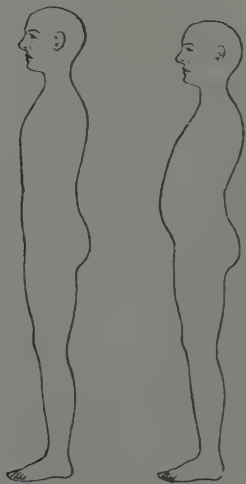

FIG. 151.—Good and bad postures.

Prevention of Malnutrition.—The most elaborate method of dealing with the problem of malnutrition in children is the one outlined by Dr. William R. P. Emerson and used in American schools, where special **Nutrition Clinics** have been established. The following is the procedure:—

(1) The child's nutrition is assessed in the manner given above.

(2) A complete personal and family history is obtained of the child.

(3) The home surroundings of the child are investigated from the point of view of diet, habits as regards sleep, &c.

(4) A complete physical examination is made to detect any cause of malnutrition, such as enlarged tonsils, adenoids, anæmia, tuberculosis, &c.

(5) In suitable cases, X-ray examination and the Wassermann test are made to detect obscure tuberculosis or congenital syphilis.

When the cause of the malnutrition is detected, a programme

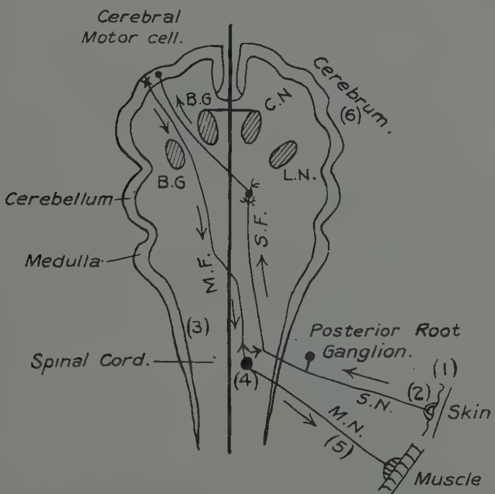

FIG. 152.—Diagram of the nervous system. The numbers correspond to the numbers in the text on p. 652. B.G. are the basal ganglia consisting of C.N., caudate nucleus, and L.N., lenticular nucleus. M.F. = motor fibre; S.F. = sensory fibre; M.N. = motor nerve; S.N. = sensory nerve.

is mapped out to cover the needs of the child at home and at school and the co-operation of the parents, teacher and child is secured for the purpose of correcting the faulty health habits. Physical defects are removed, proper and regular feeding is encouraged. Fresh air at home and at school is secured; and fatigue prevented by means of sufficient sleep, &c. The progress of the child is watched from time to time and the spirit of competition among the children in the same nutrition class contributes a great deal to the success of the work. The child gaining

most is given a special medal and is placed first in the class: the others follow in order of their weight. Care is taken also to prevent overweight. The results have been very good.[1]

Mental growth. — The nervous system may, very aptly, be compared with a telegraph system. It consists of (see fig. 152):—

(1) *Receiving stations*, viz., the sense organs, which receive tactile, painful, gustatory, visual, auditory, olfactory, articular (spatial) impressions.

(2) *Afferent wires*, viz., the sensory nerves which carry these impressions to

(3) *The district office*, viz., the spinal cord, where

(4) *The clerks*, viz., the nerve cells, receive and transmit the impulses or messages along,

(5) *Efferent wires*, or motor nerves to muscles or glands.

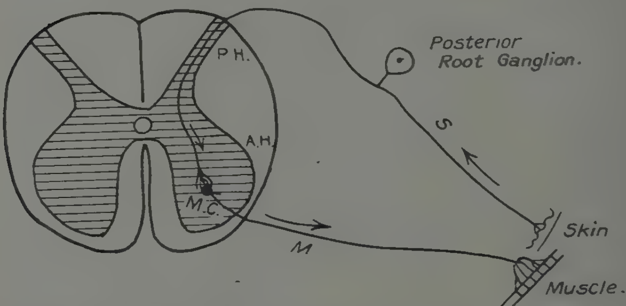

FIG. 153.—Diagram of a reflex arc. A.H. = anterior horn; P.H. = posterior horn; M = motor nerve; S = sensory nerve; M.C. = motor anterior horn cell.

In addition there is also

(6) *The head office*, i.e., the brain, which supervises the work of the subordinates, and which can originate movements which lead to actions.

The brain is the seat of intelligence, consciousness and will. It interprets the various sensory stimuli into sensations of touch, pain, taste, sight, hearing, smell, position in space, &c. In addition, the brain controls the movements of circulation and respiration and the balancing of the body. Each side of the brain receives impulses from and sends impulses to the opposite side of the body.

[1] William R. P. Emerson, "Nutrition and Growth of Children."

Hence certain movements—called **reflex acts**—can occur as the result of sensory stimuli received through sensory nerves by the cell stations in the spinal cord which reflect these stimuli along motor nerves, without the intervention of the brain, but no voluntary movements can take place, nor can various sensations be appreciated in its absence. The nerve path along which a reflex act is carried out is called a **reflex arc.** (See fig. 153.)

Each nerve fibre resembles an electrical cable, or telegraph wire, in that it consists of a central core (or axis cylinder, or **axon**), which conducts the impulse, and an insulating covering (the white fatty myelin sheath, or medulla), which prevents leakage of the current. (Fig. 154.)

[The author has shown that the relative thicknesses of the axon and of the medulla are exactly those which one would expect mathematically on the supposition that the latter acts as an insulator and allows the impulse to travel along the nerve at the quickest possible speed.[1]]

Inside the brain there are some thousands of millions of cells, each of which is connected not only with the axon of its sensory or motor fibre, but also possesses numerous other fibres, called association fibres, which establish communication between one cell and another.

Each cell, together with its various fibres, constitutes a *neuron*.

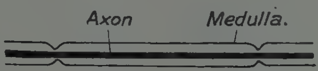

FIG. 154.—Diagram of a nerve fibre.

Brain Centres.—It was first suggested over a hundred years ago by Franz Joseph Gall and his pupil, Johann Caspar Spurzheim,[2] as well as other leaders of phrenology, that certain parts of the brain are specially concerned with certain functions. This was first experimentally shown to be true by Fritsch and Hitzig of Germany, and David Ferrier of this country. These specialized parts are called centres.

Myelinization and Function.—It is now definitely established that although an axon can function before it becomes covered with its myelin sheath, yet its function is imperfect until this

[1] See W. M. Feldman, "A Mathematical Point in Connection with the Function of the Myelin Sheath," *Proc. Physiological Society*, Jan. 20, 1923.

[2] Gall and Spurzheim, "Anatomie et Physiologie du Système Nerveux." Paris, 1810-19.

myelinization occurs. Different systems of fibres in the brain and spinal cord become myelinated at different periods of ante-natal and post-natal life, and Flechsig, in 1876, from his microscopic studies of the dates of appearance of the myelin sheath concluded that the development of the functional power of any brain area was contemporaneous with the myelinization of the nerve fibres connected with that area. In this way he ascertained the order in which various cerebral functions become firmly established.

Conditioned Reflexes.—Reflex acts, like those of blinking of the eyelids, sneezing, the watering of the mouth at the sight or smell of food (or in babies the opening of the mouth at the sight of food), the removal of the foot when the sole is tickled even when a person is asleep, &c., are called ordinary reflex acts. There are certain other acts which are called *conditioned reflexes*, in which an ordinary reflex act is in special ways hitched on, as it were, to the fibres S.F. and M.F., connecting the spinal centre (4) of the ordinary reflex arc with the brain (see fig. 152). For example, if during the process of stimulating the sole of the foot a tuning-fork of given frequency be made to vibrate under suitable conditions, and the process be repeated a sufficient number of times, there is an association established between the stimulus of sole tickling and the sound of that particular frequency, so that when the same tuning-fork is made to vibrate within the person's hearing the foot is automatically withdrawn although the sole is not simultaneously tickled. Such an act, which primarily originates from a specific stimulus conveyed to the spinal cord, but which is made to become associated with another sensory stimulus (called the conditioning or substituted stimulus) perceived by the brain is called a conditioned reflex. It was originally studied by Pavlov, but has recently been utilized by Krasnogorsky, in Russia,[1] and Watson as well as Florence Mateer, in America, not only in the study of the psychology of infancy, but also in the training of infants and children (see p. 673). The conditioning stimulus need not be an auditory one; it may be visual (e.g., a light of given wave length), olfactory (a particular odour), tactile, &c.

The Growth of the Nervous System.

(1) *The brain* is relatively very large at birth. This accounts for the comparatively large size of the infant's head. It grows

[1] See N. Krasnogorsky, *Amer. Jour. Dis. Child.*, xxx, 1925, p. 753; also W. M. Feldman, "Child Physiology," pp. 559 *et seq.*

most rapidly during the first 8 or 9 months, when its birth weight is doubled. By the end of the third year its birth weight is trebled, but between then and puberty its weight increases to no more than three-and-a-half to four times its birth weight. In other words, about 33 to 40 per cent. of the total increase in brain weight from birth to maturity occurs during the first 9 months, and 80 per cent of the total increase occurs by the end of the third year.

In addition to growth in weight, the brain also grows in structural complexity. According to Donaldson, the number of nerve cells in the brain, viz., about three thousand millions, is complete at birth, and cerebral growth consists, not in a multiplication of these cells, but in an increase of their size to any degree from zero up to 500,000 times. But although the cells do not increase in number, the fact that some of them remain small and undeveloped, even in the adult, shows that probably no human brain has ever realized all its potentialities.

As the cells grow, their association fibres also undergo progressive development, and the cortical grey matter must therefore grow in superficial area to accommodate them. To do this without a corresponding increase in the size of the skull, the brain folds itself into layers. Hence the brain, which in early fœtal life is smooth, becomes more and more wrinkled and convoluted as cerebral growth proceeds. It has been estimated that while the superficial area of the brain is about 100 square inches, the total area of the grey matter when the convolutions are spread out, is about 340 square inches. In idiots and imbeciles the brain is smooth and small and the skull is consequently also small (microcephaly). But large size of the head does not necessarily denote great cerebral development; it may be due to rickets, or may even be associated with imbecility due to hydrocephalus (water on the brain). Further, the axons coming from the cerebral cells are not all fully myelinated at birth, and part of the rapid increase in weight during the first few postnatal months is due to the deposition of the myelin.

(2) *Order of Myelinization.*—Flechsig showed that at birth myelinization is complete in: (i) The peripheral nerves (sensory and motor) which carry impulses to and from the spinal cord (myelination starts in the fifth fœtal month); (ii) The paths which convey impressions from the skin, the mouth and the sense organs to the brain; (iii) Those brain fibres which subserve respiration, circulation, sucking, swallowing and other immediately essential functions of life.

After birth, the pyramidal tracts which convey voluntary motor impulses from the brain are not completely myelinated till the third month or even later. Hence we see that at birth the only movements that are present are reflex in character. In other words, the newborn baby is a "spinal animal."

During infancy and childhood voluntary motor actions of different degrees of complexity develop. The association fibres which have to do with higher mental activities, such as thinking and reasoning, keep on developing throughout the period of growth and probably even after growth has ceased. Intellect is lodged in the frontal lobes. These develop rapidly at about the sixth year of life, when the forehead becomes more prominent.

(3) *The Cerebellum*, which has to do with co-ordination of movements of the various parts of the body and with the maintenance of equilibrium, is very small and undeveloped at birth. It grows rapidly during the first year as these functions are acquired, and the occipital region of the head which is flat at birth swells out in consequence.

(4) *Cerebral Excitability*.—Numerous observers have shown that it needs a higher current to evoke muscular movements from brain stimulation in newborn than in older animals.

(5) **Relation between Cranial Capacity and Intelligence.**—On p. 630 I have given a table showing the cranial capacity at various ages from birth onwards. Whilst this gives one an idea of the size of the brain at each age, it affords no information of the intelligence of the child. R. W. Reid and J. H. Mulligan[1] investigated the relationship between cranial capacity and intelligence in 449 Scottish students. They estimated the capacity from the height, length and breadth of the head, and the intelligence was assessed by the number of marks obtained in the various subjects of their professional examinations. They found that there was no correlation between these two measurements and they therefore agree with Pearson and Macdonell that anthropometric examination of a person's head is no guide to his intelligence.

Psychomotor Development.—At birth the baby's movements are purely automatic. It can cry and suck—and it also has a very powerful grasp, so that if made to grasp a stick held horizontally the baby can hang on to it with one or both hands and support its own weight—a fact of some evolutionary interest

[1] *Journ. Roy. Anthr. Inst.*, liii, 1923, p. 322.

(fig. 122, p. 474). This grasp gradually weakens as the baby gets older. Within a few weeks the baby shows some sign of pleasure when a brightly coloured object or light is shown to it. It also manifests evidence of memory, so that when held in the position usually assumed in feeding, it makes movements with its mouth expressive of the desire to be fed (conditioned reflex). By the end of the third month it can generally recognize its mother. It will also wink if a finger is brought close to its eyes.

At 4 months it can hold its head erect. It can also do some purposive act, such as looking for an object that is shown to it.

At 6 to 7 months, the baby can sit up erect when supported. It can reach an object; can laugh and can play with a rattle. Dentition begins about this time.

At 9 months, it can sit up unsupported, and soon after can stand up with some support.

At 10 to 12 months it can stand up alone.

At 12 to 15 months, it begins to walk and to utter some words.

Whilst considerable variations may occur in perfectly healthy children, great delay in the development of these acts is due to some physical or mental cause, such as rickets, general malnutrition, paralysis (cerebral or spinal, or due to some bony or muscular lesion), mental deficiency (e.g., mongolism, &c.) Hence, by noticing the age at which an infant acquires the ability to perform certain acts, it is possible to estimate to some extent the degree of his muscular and mental development. If speech is not developed by the time the child is three years old, mental deficiency may be suspected—provided that hearing is not at fault, or the child is not suffering from debility due to some prolonged illness. In the latter case, speech will come when the baby gets stronger. If deafness is the cause, then its relief will result in the acquisition of the speaking faculty.

Development of the Senses.

Taste.—The sense of taste is well developed at birth.

Smell.—The sense of smell is probably well developed at birth. It is even said that a new-born baby can distinguish, by smell alone, various kinds of milk.[1]

Hearing.—For the first few days, hearing is almost entirely absent (see p. 532). It rapidly improves and soon becomes very acute.

Sight.—Vision is very feeble at birth because the visual centre in the brain is not yet fully developed. During the first

[1] See "Child Physiology," p. 237.

few weeks a strong light causes discomfort, and before six weeks the baby is said not to be able to focus its eyes. It begins to recognize objects after the third month.

Touch is strongly developed at birth.

The Influence of Physical upon Mental Growth.—Although the saying "Mens sana in corpore sano" is not universally true it is generally so. Cronk found that mentally backward children suffered half as much again from malnutrition and naso-pharyngeal obstruction and three times as much from defective hearing as normal children. Quite apart from the possibility that physical and mental growth may both depend upon the same factors, it is clear that a physically defective child has not the same opportunities for schooling as its normal companion. Other observers find a similar relationship between physical and mental growth and add defective vision as a cause of mental backwardness. Shrubsall finds the correlation coefficient between visual acuity and school standard of the child at different ages between 9 and 12 years, for the two sexes, to fluctuate between $0{\cdot}113 \pm 0{\cdot}007$ and $0{\cdot}159 \pm 0{\cdot}007$, which, though small, is significant, being more than three times its probable error. E. G. Habakuk[1] finds that mental and physical development go hand in hand, so that the best children mentally are the best physically. The following is a modified table of his findings in the case of 324 boys, 10½ years old.

TABLE CCXXV.

School class of child	Mean weight (kg.)	Mean height (cm.)	$\frac{\text{Mean weight}}{\text{Mean height}}$
I	23·89	122·7	·1948
II	26·15	126·1	·2074
III	27·28	128·4	·2124
IV	28·51	131·4	·2170
V	29·50	133·5	·2210

From this table we see that the weights, heights and $\frac{W}{H}$ ratios of the children of the same age increase as we pass along the school standards. Moreover the rise in $\frac{W}{H}$ ratio shows that per unit of length, children of the higher standards are heavier

[1] *Journ. Hyg.*, xxv, 1926, p. 295.

than those in the lower. In other words weight has a more important influence on mental development than has height.

Relation between Environment and Intelligence. — L. Isserlis[1] finds that there is distinct correlation between the intelligence of school children and their environment, whether measured by the economic position of the parents, by the care taken at home, or by the clothing of the children. The partial correlations for constant age are of the order three to four and five to six times greater than their probable errors. Karl Pearson,[2] however, disputes the significance of Isserlis' findings.

[1] "The Relation between Home Conditions and the Intelligence of School Children." Med. Res. Council, Special Report Series, No. lxxiv, 1923.

[2] "Biometrika," xv, 1923, p. 161.

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CHAPTER XXVI

THE MENTAL HYGIENE OF EARLY LIFE

> "To aid thy Mind's Development—to Watch
> Thy Dawn of little Joys—to sit and see
> Almost thy very growth—to view thee catch
> Knowledge of Objects—Wonders yet to thee!"—Byron.

The Psychology of the Child.—Haeckel's law of biogenesis states that "every embryo in the process of its development goes through a series of changes which represent the permanent forms of other animals lower down in the evolutionary scale." This law, which was announced in respect of the morphological development of man, also holds good in regard to his mental development. The child "climbs up its own genealogical tree" not only in the morphological but also in the psychological sense. The truth of this can be realised by watching children of various ages at play. During early childhood the little boy or girl, whether rich or poor, loves to play with sand. The children of the well-to-do will build sand castles at the seaside or in their gardens; those of the poorer classes will make mud pies whenever they get the opportunity. This instinctive love of earth is believed to be reminiscent of the period in the development of the race when man lived in caves. Other games at about the same age or a little later are those of adventure in which secrecy forms a part. They will play at hiding, pirates, big game hunting, &c., recalling another stage in racial development, when our ancestors lived at war with their neighbours, man or beast, and when they sought protection from their foes by concealing their movements. Later on games of ceremonial and of construction occupy the child's mind. The boy will play with mechanical toys, build houses, boats and other structures, whilst the little girl will play with her dolls and cherish them with the love that a mother bestows upon her children. At a later stage still the child elicits an interest in organised games such as marbles, cricket, football, which represent the final stage in the sociological development of the race, where

people have to live in social intercourse as well as in friendly rivalry with their neighbours.

It is necessary to recognise these various stages in the psychological development of the child in order that we may guide it and help it along in the right way.

The Child's Emotions.—By an emotion is understood the feeling evoked in the mind in response to some external stimulus. Emotions are of various kinds, such as pleasure, disgust, fear, anger, love, hate, &c. The most important emotions in early childhood are those of fear, anger and love, as each of these plays its part in the formation of character. All these emotions are to a certain extent instinctive, but are also in part the result of past personal experience.

Fear.—Children are instinctively afraid of the dark, of strangers, loud noises such as thunder, &c. This kind of fear is an instinctive emotion hereditarily bequeathed to us by our ancestors, who were constantly at war with their neighbours and necessarily had to avoid strangers and darkness. Such fears gradually disappear as the child grows older and is in a position to realise that the danger is only imaginary, but he can be helped along to overcome it by tenderness, by explanation, and above all by a demonstration of the absence of fear on the part of his parent, nurse or guardian. To a certain extent, however, such instinctive or inborn fear is as serviceable to the child as it was to our ancestors, inasmuch as it protects him from danger.

[That instincts are inheritable has been recently demonstrated by Pavlov, when he showed that conditioned reflexes in dogs are transmitted to future generations. See p. 674.]

The fear which is based on experience, e.g., the child's fear of a dog because of having been once pounced upon or bitten by one, also has a protective value. The child's vivid imagination and capacity for imitation are also sources of fear. A horrible story, or the sight of his mother in a state of fear, will immediately call forth this emotion in the child. Hence great care must be taken in the selection of the kind of story that is told to a child. The policeman or the doctor should not be used as a sort of bogeyman to frighten a child, as is occasionally done by a foolish mother or nurse; he should be trained to regard them as his friends who will help him out in a time of trouble.

Anger.—This is the result of some annoyance, such as physical discomfort, fear, or deprivation. This emotion is accompanied by the instincts to scream and strike. Fits of temper are exceed-

ingly common in childhood because they represent a stage in phylogenic development or the development of the race when uncivilised man was violent and explosive. Such outbursts of temper should not be countered by loss of temper on the part of the nurse, but should be treated with tact and sympathy. He should be left alone for the time being, and, when the fit of temper has passed off, should be made to realise that not only was there no real cause for such an exhibition, but that he gains nothing by that kind of conduct. At the same time one should, as far as possible without spoiling the child, avoid any real cause for such display of temper. With the process of his mental evolution his instinctive fears will gradually vanish.

Repression.—Each emotion requires an outlet. Fright, for instance, will find its outlet in a fit of crying, which forms a safety-valve for the pent-up nervous tension. This should therefore not be peremptorily checked or repressed, otherwise the delicate nervous system might suffer considerable damage. For, although the suppressed emotion may be forgotten for the time being by the conscious mind, it may remain latent in the subconscious mind and may manifest itself in some form of neurosis during a later period of life. It is in such forms of neurosis where psycho-analysis enables one to discover the nature of the repressed emotion and cure the condition.

Provision of an Outlet for Emotion.—The proper way of dealing with a child who has had a sudden shock such as a fright is to endeavour to transform the energy of crying into some other form of energy, by soothing it and trying to divert its thoughts into other channels. When the child is able to speak one can induce it to tell the incident to the mother or nurse. If this is done the child will feel that he can always find sympathetic ears into which he can pour out his tale of woes, so that should he receive another shock, he will remember not to go into a fit of crying but will hurry, probably with tears in his eyes, to tell his experience to people ready to listen.

But whilst sudden repression is bad, it must be remembered that the whole system of training must necessarily entail a great deal of exercise of inhibitory control or gradual repression, otherwise the child will grow up into a savage unfit to mix in decent society.

Psychological Peculiarities of Childhood.—In addition to great *physical activity* associated with early life (see p. 666), there are several other mental peculiarities characterising this stage of the child's development.

Curiosity or Inquisitiveness.—This trait is merely a natural appetite after knowledge which should within reasonable limits be encouraged. "The human mind," says W. E. Castle, "is characterised above all else by curiosity, the source of our wisdom as well as of our woes. This fact the ancients portray in the tale of Pandora's box. . . . We demand a reason for everything, and if none is forthcoming from an outside source we construct one for ourselves. . . . This is the attitude of mind of the child whose perpetual 'Why' and 'What' are so distressing to perplexed parents."

The child who takes his engine to pieces to see how it works is simply carrying out a natural and perfectly praiseworthy impulse, and all toys given to children should be such as to enable them to satisfy their legitimate curiosity without any injury to themselves or undue expense to their parents. It has been well said that a child that is not curious is a very curious thing.

As soon as the child begins to talk and can easily express himself in words, he will begin to attempt to satisfy his curiosity by asking questions, which gradually become more and more searching and embarrassing, e.g., questions about God, about death, and on matters relating to sex and the origin of life. These must not be shirked or evaded, but should be answered patiently, delicately, and above all truthfully. It would be impossible to go into these matters in detail in this book; suffice it to say that the **questions relating to sex and the origin of life,** which are, of course, the most difficult to answer, should to a certain extent be forestalled by giving the child, of suitable age, a few elementary demonstrations and lessons in nature study, when the process of reproduction in plants might be explained to him and a few words might be given on planting seeds in the soil. Gradually the child may be given to understand that animal or human reproduction consists of a somewhat similar process to that prevailing in plants and in the sowing of seeds, but at the same time it must be impressed upon the child's plastic and receptive mind that reproduction is one of the most sacred phenomena in nature, which should never be referred to unnecessarily nor spoken of with levity. It should be explained to him that when that mysterious power which we call God created the world and all the living things that are in it, He created them in such a wonderful way as to render it unnecessary for Him to go on creating them afresh each time, but that He has endowed them with the power of themselves continuing

the process of creation, and in this way delegated to the human being the marvellous power of creating man in the image of God, and that this is what is meant by the blessing, "Be fruitful and multiply and replenish the earth."

At a later stage the whole subject of elementary physiology, including that of reproduction and of fœtal nutrition, should be taken up by a competent teacher who will deal with it in the same matter-of-fact way as with the other bodily functions.

Imagination and Imitation.—Children are very imaginative and imitative, because the inhibitory control of the higher brain is still imperfectly developed. Their vivid imagination unconsciously makes them tell untruths. They may see a couple of cats in the garden, and come back and say they have seen thousands of cats. I have recently been told by a child at the seaside that she "could not see the sea for the bathers," when I found that there were seven people in at the time. Such exaggeration should be gently and lovingly rectified. It must be remembered that children are more amenable to love and tenderness than to reason, because emotion is developed before logic. Their imitativeness makes it very necessary not only to isolate them from other children suffering from certain functional diseases such as stammering, habit spasm, chorea, &c., but also that they should be placed in surroundings with right influences. Hence a refined and cultivated home will do more for the mental development of the child than the best school. Further, it is his imitativeness and lack of critical faculty that renders the child so amenable to suggestion, a fact which is to be carefully taken into account in the training of children.

The Child's Point of View.—It must be realised that children's points of view are not the same as those of adults. For instance, a baby estimates size by his own standard: objects much larger than himself look gigantic to him. Hence he may get frightened at comparatively small objects. Further, a child's actions may be very slow and clumsy because of lack of practice and past experience. Hence, those in charge of young children must be patient with them. They must be allowed to dress slowly until they become proficient, and, above all, eat slowly. Another thing is that their power of concentration varies inversely with their age, and hence the duration of mental exercises should be short and the intervals between them long.

Untruthfulness.—Young children are very apt to tell lies, for several reasons:—

(1) *Imagination* (see above).

(2) *Imitation*. The child may have heard his parents or nurse say something untruthful. The cure for this is to remove the cause.

(3) *Fear*. The child may be afraid to own up to having done some damage, &c., for fear of punishment. The cure lies in removing the fear and developing the child's moral courage.

(4) *Laziness*. He may tell a lie to save himself some trouble. For instance, he will say that he has washed himself for his meal in order to save himself the trouble of washing. The cure is to make the child see that it does not pay, by sending him to rectify the omission.

Mental Education.—From the point of view of mental development, in the same way as in the case of physical development, the new-born baby is a complete but unfinished bit of humanity, and the same principles that guide us in promoting healthy growth of the baby's body should also help us to enable its brain to develop in the best possible manner. We do not now swaddle babies or put growing girls into corsets with the idea of straightening their limbs and of supporting their spines, but, on the contrary, we allow their muscles plenty of scope for free movement and development. Similarly, we are gradually beginning to realize that equal freedom must be given to the child's sensory and mental faculties from the earliest possible moment, so that they may have the best opportunities for developing themselves in the best way.

Infancy and the early years of childhood, when the child's brain is still not only "a white paper unscribbled with observations of the world," but is also very plastic and impressionable, are the best times for laying the solid foundations of individual character.

Principles of Mental Training.—The first thing to remember is that mental development must come *mainly* through the child's own effort. The child that is not allowed to touch or handle things or do simple tasks for himself lest he should hurt himself grows up with the corresponding portion of his brain insufficiently developed, and unless very quickly remedied he will grow up lacking in experience, initiative and self-reliance. On the other hand we must, of course, take care that in the process of his acquiring experience he does not harm himself unduly, e.g., by handling dirty, sharp, or very hot objects.

The problem of mental training in the early years of life is so

to adjust the child to his natural environments as to secure the least amount of unnecessary repression.

Physical Activity.—The most noticeable feature of normal infancy is physical activity. If a child lies awake in its cot and is perfectly still, it is practically certain that there is something either physically or mentally at fault with it. It is through this activity that the baby gains experience of its environment through contact with various objects. Its own skin is a mass of sensitive organs conveying tactile, including stereognostic and thermal impressions, as well as those of pain and pleasure, to the brain, and the constant reception of these, as well as of visual, gustatory and auditory stimuli, helps the cerebral cells and fibres concerned in the appreciation of these various sensations, to develop. The various movements of its joints also train the child in spatial perceptions.

As the child grows and begins to sit up or walk, his activity grows, and busy, impatient mothers or nurses are apt to order the very active child to sit still, or not to do this, that, or the other thing. Such interference with the child's normal activity, especially when he is engaged upon some new task which he is trying to learn, and when his whole nervous system is in a state of high tension, is apt to have the same effect upon it as trying to keep the steam in a kettle by stopping the spout. A nervous explosion may result and the child's nervous mechanism may suffer considerable damage. The nurse or parent should therefore be sparing in the use of her "Don'ts." For not only is frequent mental repression harmful, but the word "don't" may actually convey an idea to the child which we do not wish to put into his mind. Thus, a nurse on leaving a child alone in a room may tell him not to touch the fire in her absence. This injunction may call the child's attention to the fire, and his natural curiosity may prompt him to ascertain why he must not touch it. The wiser thing is to have a guard in front of the fire, and leave the room without mentioning it. Of course, there are times when instant prohibition is necessary, and the child must be trained in implicit obedience, but we must use such prohibition very economically.

Training the Senses.—The five senses—touch, sight, hearing, taste and smell—have been picturesquely described by Bunyan as "the gates of the City of Mansoul."[1] These gates to the soul or mind are not of equal importance. *Taste and smell*, for instance,

[1] Compare Aristotle's dictum: "Nihil in intellectu quod non fuerit prius in sensu."

though the first to develop because they are the senses most necessary to life, are not so significant as the others. Through the exercise of the gustatory and olfactory senses the child learns to differentiate between the various flavours and odours. A child has a natural instinct for sweet flavours, as has been proved experimentally by noticing the effects of sweet, bitter and other flavours on very young babies, when it was found that sweet always elicits an expression of pleasure. When the desire for sweet things manifests itself in the child it should be satisfied wisely—but it must be remembered that this, like other natural appetites, needs training in order to prevent abuse. *Hearing* develops very slowly, and as we have seen, it usually takes two or three days for this sense to manifest itself. It is through this sense that the child learns to understand and to speak, and from very early infancy the child can be trained to appreciate rhythmical sounds. *Sight*, like hearing, develops slowly. The baby soon begins to enjoy coloured objects, and training in the proper appreciation of art and beauty should begin with the tasteful decoration of the nursery (see p. 324).

The sense of touch, however, is the one which, as we have seen, enables the young infant to acquire the greatest amount of knowledge and experience. Everything that comes a baby's way goes immediately to its mouth. If it is pleasant it is sucked, and if it is nasty it is rejected. In this way it learns to discriminate between pleasant and unpleasant tastes.

The enormous developmental and educational potentialities that are latent in the tactile sense are illustrated by the history of Helen Keller, an American girl who, at the age of two years, became deaf and dumb and blind, as well as deprived of the sense of smell through an attack of scarlet fever. Under skilled guidance, however, to excite her curiosity, she learned, by the sense of touch alone, not only to read and write Braille but also to speak, so that she went through a regular school, college and university training, took a mathematical degree and wrote several books.

The Swiss educational reformer, Johann Heinrich Pestalozzi (1746-1827), was the first to indicate the possibility of developing all the faculties of a child through the exercise of its senses. He did not, however, develop his scheme, and it was left to his pupil, the German educationist, Friedrich Wilhelm August Froebel (1782-1852), to apply the method practically to quite young children. Froebel created the Kindergarten (the Garden of Children) system, called after the school he started in the Thuringian Forest. His plan consists in training children by their own activity

FIG. 155.—Children gardening.

FIG. 156.—Montessori Methods. Children washing.

FIG. 157.—Children serving dinner.

FIG. 158.—Montessori Methods. Children washing dolls' clothes.

expressed in the form of pleasant play. About a quarter of a century later Edward Séguin, a French refugee in America, introduced a system of teaching idiots by the method of simple passive mechanical arm and hand movements, and by the patient training of the sense of touch. Maria Montessori applied Séguin's method to the training of normal children.

The Montessori System is divided into three parts:—

(1) *Motor Education*, which consists in giving order and direction to the child's unco-ordinated movements, leading him to do those actions which he is aiming to do. These muscular movements have reference to walking, handling objects, personal care (dressing and undressing), domestic work, gardening, gymnastics, rhythmic movements, &c. (Figs. 155-158.)

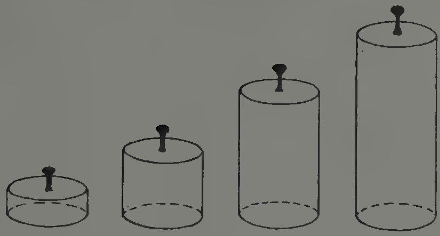
Thus, for the purpose of teaching dressing and undressing there is a collection of frames to which are attached pieces of stuff, &c., which can be buttoned, hooked or tied together. The teacher performs on these frames the various necessary movements involved in fastening or unfastening our clothing, one act at a time, and very slowly, in front of the child. The child is then given the frame, and is encouraged to repeat these actions for himself. In this way they soon learn to dress and undress themselves. Similar methods are adopted for teaching other movements, such as washing, gardening, balancing and rhythmic movements.

(2) *Sensory Education.*—The child is given rows of cylinders, differing either in height only or in diameter only, or both in height and diameter (see fig. 159). One row of ten cylinders of the same kind is given at a time, and the exercise consists in placing them in the appropriate hollows in a block of wood. The child between 2 and 3 years soon finds that one cylinder is either too wide or too narrow, too long or too short for the hole into which he tries to insert it. By repeated trial and error he soon learns to fit all the cylinders into their appropriate holes. In this way he learns, from his own mistakes and unaided efforts, to appreciate differences in dimension, to observe, to form judgments and to reason.

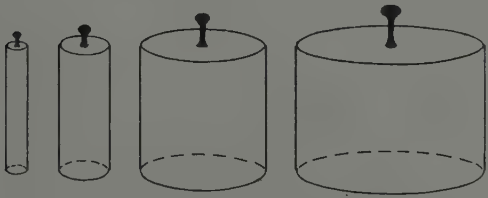
When the child has practised and become efficient with the cylinders, he is given other geometrical forms, such as cubes and prisms, to arrange in order of magnitude. Other exercises have been devised to train the sense of touch, temperature, as well as the appreciation of colour.

(3) *Language and Writing Education.*

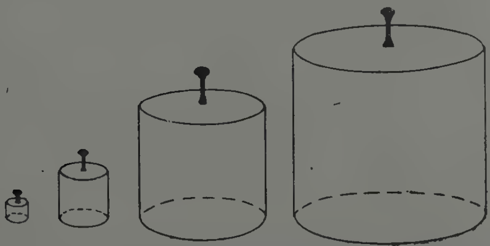
The main principle of the Montessori system is, as has been seen, that of auto-education—in other words, education without guidance or interference by the teacher. The teacher mainly



(*a*) Cylinders of same diameter but of different heights (i.e., differing in one dimension only).



(*b*) Cylinders of same height but of different diameters (i.e., differing in two dimensions).



(*c*) Cylinders of different heights and diameters (i.e., differing in three dimensions).

FIG. 159.—Training of Senses.

shows how a certain thing is to be done, and leaves it for the child's innate instincts of curiosity and imitation to repeat the exercise and learn from his own mistakes. The system is certainly a very attractive one, but the fact that the teaching is done without guidance renders it applicable only to children who are mentally perfectly normal, but, as Sir James Crichton Browne, in a criticism of the system, rightly says[1]: "In the human garden there are weeds, briars, and thistles, as well as lilies and roses, and morbid hereditary tendencies as well as pathological bacteria are at work." For such children guidance and interference from outside are absolutely necessary to enable them to develop their minds in a normal manner. But even normal children are in need of some guidance, for, as Rudyard Kipling puts it:—

"Gardens are not made
By saying, 'O how beautiful!'
And sitting in the shade."

Psychometric Tests.—It is now possible, by means of Binet's tests or their modifications, to discriminate accurately between the normal, clever and backward child at a very early age. An outline of these tests has been given in Chapter VII (pp. 224 *et seq.*). These tests show that whilst the vast majority of children are of course neither extremely clever nor extremely stupid, the percentages of children belonging to either extreme are about equal. In other words, the distribution of mental capacity follows that of the normal frequency curve. (See Ch. III, p. 86 *et seq.*)

Education and Instruction.—It is important to realise that education and instruction are not synonymous terms. The object of the former is to train the mind in such a way as to enable it to think clearly and to develop a critical faculty. The object of instruction is merely to impart information. The tendency in the case of children is to give them too much instruction and too little education. As Mark Twain said: "Don't let your son's school interfere too much with his education."

Habits.—The constant repetition of any act results in the formation of a habit. This is particularly the case in children, and the younger the child the more easily is the habit acquired,

[1] "Child Study and Pathology," *Child Study*, 1913.

because the paths in the nervous system of the child are still plastic and untrodden, so that a very few repetitions of the act will be sufficient to leave a lasting impression. It must be remembered that it is as easy to train a child into good habits as into bad ones, and that it is as difficult to get out of a bad habit as of a good one. Hence the training of a child into good habits should begin at the earliest moment, i.e., immediately after birth. The first habits to be inculcated at this early age are regularity of feeding and of evacuation of the bowels (see p. 318). As the child grows older, the food habits must be enlarged in scope so that the child is accustomed to eat any food that is offered to it without coaxing. It must also be made to acquire habits of kindness, unselfishness (and therefore lack of jealousy), restraint and self-control, self-reliance, accurate observation, personal cleanliness, punctuality, truthfulness and courtesy.

The Use of Conditioned Reflexes in Child Psychology and Training.—Supposing it is desired to ascertain whether a baby or child can distinguish colours, and if so, what are its limits of spectral sensitivity. A conditioned reflex (see p. 654) is established with a colour of given wave-length as the conditioning stimulus. A colour of different wave-length is then presented to the child, and if, but not unless, he is able to distinguish it from the original colour, the reflex will appear. Again, the sensitivity to touch, heat, or cold, &c., of any part of a baby's skin can be ascertained by making any of these stimuli, conditioning stimuli, and after the reflex has become established applying the same stimulus to any portion of the skin whose sensitivity is to be investigated. Good and bad habits can also be acquired by associating any particular behaviour with some particular conditioning stimulus. Thus by holding out a baby over a receptacle at times when a movement of the bowels is found to be taking place naturally, a conditioned reflex is in time established between the sight of the receptacle and the reflex act of defæcation, thus ensuring clean habits even in very young babies. Similarly, allowing baby to sleep always in a dark room will render it unable to fall asleep in a lighted room, but by varying the illumination of the nursery from time to time, the baby can be trained to go to sleep under different conditions of lighting. If the same flavouring agent is always given to a child to conceal the taste of a nasty drug, an association will be established between the drug and that flavouring agent, so that a dislike, or even a feeling of disgust, will be established in the child for that particular flavour. The author,

for instance, dislikes the taste of peppermint because in his childhood it was often used as a vehicle for the administration of castor oil. Such examples can be multiplied almost indefinitely and their value in child training will be easily appreciated.

The Inheritance of Conditioned Reflexes.—Pavlov has recently shown that the number of repetitions of the conditioning stimulus necessary to establish a conditioned reflex becomes less and less with each generation. Thus he established a conditioned reflex for food (i.e., salivation) in a litter of mice by sounding a bell coincidently with the exhibition of food, 300 times. The next generation formed the same habit of reflex salivation after 100 repetitions only; the third generation after thirty repetitions; the fourth generation after ten repetitions, and the fifth generation after five repetitions only. This shows that conditioned reflexes are inheritable and may become established as instincts. If this also holds good for man, it would mean that good or bad habits or moral tone tend to be transmitted from parents to offspring, so that a refined home has a good influence upon the child, not only environmentally but hereditarily.

The child should be trained along an ascending scale of acts of endurance and self-denial, and deeds of kindness which involve subordination of self to the interest and benefit of others. He must be trained to be able to withstand temptation, even if left alone with some chocolate, jam or other dainty of which he is particularly fond, and he must learn to give up, without any compunction, a favourite toy to another child who is in greater need of it than he. In this way self-control and chivalry will gradually be acquired as a habit, and the child will, at a later period, be able to withstand the temptation to gratify illicit impulses (sexual or other).

The teaching of such good habits will thus lay an excellent foundation for the moral development of the child, which after all is as important as his proper physical development. For in the words of the poet (Sir A. Hunt):—

> "What is Beauty? Not the show
> Of shapely limbs and features. No!
> These are but flowers
> That have their dated hours
> To breathe their momentary sweets, then go.
> 'Tis the stainless soul within
> That outshines the fairest skin."

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CHAPTER XXVII

ADOLESCENCE OR PUBERTY

"On Cope's view the influence of environment in producing acquired characters transmitted by heredity is greatest on the soma during adolescence. At any rate, for those prophetic souls interested in the future of our race and desirous of advancing it, the field of adolescence is the quarry in which they must seek to find both goal and means."—G. STANLEY HALL.

ADOLESCENCE, or puberty, is the transition period between the stage of childhood and that of the full-grown adult. It commences at the age of about 14 to 15 in boys and about one year earlier in girls and lasts ten years, when the individual becomes a mature man or woman.

Characteristics of Puberty.—The period of adolescence is accompanied by many physiological and psychological changes. The most characteristic change is that the sexual functions are established in both sexes, so that the individuals become capable of becoming parents. During this period also the body shows a marked increase in general growth, although the rates of growth in the different organs or parts of the body are not uniform. Hair first appears in the pubic region, then in thé axillæ, and later on the face, in the case of boys. Menstruation in girls is the first, and by far the most important, sign that puberty has appeared in them, and the breaking of the voice heralds its first onset in boys.

Puberty literally means the state of becoming hairy, and the word has been applied to the stage of adolescence on account of the development of hair on the body in general, and in the pubic region in particular, at that time. Indeed the word *pubes*, which means "hair," has, on account of this association between the hairiness of the genital region and adolescence, become utilised, by transference of ideas, to mean the genital organs.

The various changes occurring at puberty are dealt with in the author's "Child Physiology." Here it will suffice to enumerate a few only which have a special bearing upon the hygiene of this period of life.

Growth.—Next to infancy, puberty is the period of post-natal life in which growth is most rapid (see fig. 149), and because quick

growth predisposes to various kinds of physical and mental disturbances, these two periods of life are "the rocks on which more than one lifeboat suffers damage" (Seham). It is true that the adolescent period is one in which the mortality-rate is lowest (see Table I and fig. 5, on p. 9), but on the other hand it is one during which the child, especially if hereditarily predisposed, is liable to a considerable amount of physical and mental ill-health. Hence the child's expenditure of energy during this period should be as little as possible, and his intake of energy as much as possible. Overstrain at school must be guarded against, especially in the case of girls, and sleep as well as proper food should be provided in abundance.

Muscles and Bones.—The muscles grow at a slower rate than the bones, and this fact is by some believed to account for the "growing pains" that occur during this period, although a more probable explanation is that such pains are of rheumatic nature. The unequal rates of growth may also account for certain bone deformities occurring during the puberty period, especially curvature of the spine.

Cardio-vascular System.—Between the ages of 13 and 15 years the volume of the heart becomes nearly doubled (see p. 636). This may give rise to a sense of fullness in the chest, making the child conscious of, and even sometimes morbidly anxious about, his heart. This fear becomes accentuated by the shortness of breath, palpitation and faintness, due to some temporary dilatation of the heart brought about by its rapid growth, as well as by sinus arrhythmia (a special kind of irregularity), common at this age. The relation between the volume of the heart and the circumference of the aorta becomes increased, so that a relatively larger quantity of blood has to be pumped through a unit sectional area of blood-vessels. This gives rise to an increased blood-pressure and a more forcible cardiac impulse which again calls the child's attention to his heart.

Blood.—Anæmic conditions are liable to set in at this period, although chlorosis, which used to be so common in girls about ten or twenty years ago, is now hardly ever seen. This may, perhaps, be the result of the more rational clothing of women, viz., light and loose frocks and no corsets, as well as their more rational mode of living with regard to exercise in the open air.

Basal Metabolism.—Notwithstanding the increased growth occurring at this period, the basal metabolism has been shown

by Benedict and Talbot not to undergo any change. This is contrary to the observations of earlier observers, especially of Dubois, who compared the basal metabolism *in the same children*, both at the onset of puberty and two years later, when he showed that the metabolism during the former period was considerably higher than during the latter period. Benedict and Talbot criticise this statement by saying that the number of observations was too small to eliminate any error due to differences in the amounts of muscular activity.

The vocal cords in boys grow from 13 to 24 mm. in length between the ages of 12 and 20, and, in accordance with the law of vibration in strings, this approximate doubling in length causes the voice to become about an octave lower. Girls also show a break in the voice, but not such a sudden one as in boys, because in them the increase in length of their vocal cords during the same period is no more than about 4 mm., viz., from 12 to 16 mm. In castrated boys the voice does not change at puberty, showing the dependence of the growth of the vocal cords upon the internal secretions of the testicles.

The ovaries are very small at birth but contain a couple of hundred thousand unripe ova. At puberty they grow very rapidly, and in the adult woman they have the size and shape of a large almond, weigh 2 oz. each, and contain no more than about 30,000 ova between them, the others having degenerated and been absorbed. Of the 30,000, no more than about 400 can become mature and escape from the ovary (one at each mid-menstrual period—i.e., fourteen days before the onset of the menstrual discharge—between the ages of about 15 and 45), and no more than about 20 can possibly be fertilised.

[The ripening and escape of an ovum from the ovary is called *ovulation*. The ovum finds its way into the Fallopian tube, which propels it into the uterus, whence, if unfertilised, it is cast out in the menstrual discharge fourteen days later. Should, however, the ovum happen to be fertilised on its way to the uterus, it becomes embedded in the wall of the uterus and the menstrual flow does not occur throughout the whole period of pregnancy.]

The Testicles.—The rapid growth of the testicles at the age of puberty is seen from the following figures, representing their weights at different ages:—

Age in years	Birth	3	6	11	14	18
Weight of testicles in grm. ..	0·5	1·2	1·25	1·7	3·9	18·1

The testicles, like the ovaries, have external and internal

secretions. The external ones are the spermatozoa, of which there are about 225 million in each ejaculation, and ova respectively, and the internal ones are manufactured by interstitial cells, called the cells of Leydig, in the testes, and by the corpora lutea in the ovaries. These internal secretions have to do with physical and mental growth as well as with the determination of the male and female sexual characters. By transplanting ovaries into castrated male animals, the latter can be converted into animals having female characteristics. Similarly, transplantation of testicles into female animals from which the ovaries have been removed, converts them into animals having the secondary male characters. Transplantation of another woman's ovary into the body of a woman whose own ovaries have been surgically removed will supply her not only with the necessary internal secretion but also with ova which might enable her to conceive and bear a child, although biologically the child would not be her own but only her foster child (cf. Castle and Phillips' experiment referred to on p. 216).

Types of Ova and Spermatozoa.—A most interesting fact regarding ova and spermatozoa is that in connection with their minute intranuclear structure. Inside the nucleus of every cell there are certain protoplasmic filaments, which, on account of their special microscopic staining characters, are called chromosomes. The number of these chromosomes is fixed for every species of animal, and there are always twice as many of them in the soma, or body cells of the animal, as in the same animal's germinal cells. In the case of the human species, the number in each germinal cell is twenty-four, and that in each soma cell is forty-eight. Of the twenty-four germinal chromosomes, twenty-three are indistinguishable from one another, but the remaining one differs from the others in being considerably shorter. This odd chromosome, in the case of the ova, is always of the shape of a short rod, and is called the x-chromosome. In the case of the spermatozoa, however, it assumes one of two shapes: in half the number of spermatozoa it is a short rod (an x-chromosome), in the other half it is a still shorter ovoid, the so-called y-chromosome. The chromosome constitution of the soma cells differs in the two sexes as follows: the soma cell of the female contains forty-six ordinary chromosomes, together with two x-chromosomes (i.e., $46 + 2x$), whilst that of the male contains forty-six ordinary chromosomes, together with one x-chromosome and one y-chromosome (i.e., $46 + x + y$).

As $(46 + 2x)$ obviously arises from the fertilisation of an ovum with an x-containing spermatozoön, thus:—

$$\underbrace{(23 + x)}_{\text{ovum}} + \underbrace{(23 + x)}_{\text{spermatozoön}} = \underbrace{(46 + 2x)}_{\text{female}};$$

and $(46 + x + y)$ must arise from the fertilisation of an ovum with a y-containing spermatozoön thus:—

$$\underbrace{(23 + x)}_{\text{ovum}} + \underbrace{(23 + y)}_{\text{spermatozoön}} = \underbrace{(46 + x + y)}_{\text{male}},$$

the x-containing spermatozoa are called female producers and the y-containing ones are called male producers.

From all this, therefore, it should follow that, other things being equal, there should be an equal chance for an ovum to be fertilised by either one or the other type of spermatozoön, and hence, theoretically, there should be as many female as male conceptions. Statistics, however, show that throughout the world there are about 105 live males to every 100 live females born every year, i.e., the **sex ratio** is 105 : 100, and if we were to take into account dead births, miscarriages, abortions, &c., the sex ratio rises to about 150 : 100,[1] or even higher. There are several possible explanations of this discrepancy between theoretical expectation and actual fact, but the most probable one is that not only is the female-producing spermatazoön more vulnerable than the male-producing one to the action of chemical agents or toxins that may be circulating in the blood (see pp. 243 and 247), but that the y-chromosome being smaller than the x-chromosome, it renders the male-producing spermatazoön lighter, more active and quicker-moving than the female-producing one, thus enabling it to reach the ovum more quickly. The result is that, although in the process of spermatogenesis there are probably produced equal numbers of the two types, fewer female-producing ones survive to compete in the race for fertilising the ovum, and these are not such good "runners" as the more numerous survivors of the male-producing type.

Sir Frederick Mott,[2] who studied the normal histology and microchemistry of the testicles from birth onwards, finds that at birth the seminiferous tubules are filled with cells which show no differentiation. Some of these cells have round nuclei; these are the spermatogonia, which during maturity give rise to spermatozoa. Others have oval nuclei; these are called

[1] See A. S. Parkes, *Journal of Genetics*, xiv, 192, p. 329.

[2] *Brit. Med. Journ.*, ii, 1919. See also Frederick Mott and Miguel Prados y Such, *Proc. Roy. Soc. Med.*, 1921-22, vol. xv, part iii, Sec. Psychiatry.

the cells of Sertoli. The testicles contain very little cytoplasm, but a very large amount of interstitial tissue, amongst which are groups of large polygonal cells—the cells of Leydig—which contain a lipochrome substance resembling the lutein of the ovaries. At 4 months the seminiferous tubes have doubled in size, but the cells of Leydig have diminished in number and contain no lipochrome. At 11 years spermatogenesis has begun, but is very slight and immature. At 15 years the seminiferous tubules are fully developed, are twice the size of those at 11 years and contain ripe spermatozoa. The cells of Leydig, with their contained lipochrome, have reappeared. The lipochrome which contains phosphorus is necessary for the nuclein required in the formation of spermatozoa. It gives the oxidase reaction, i.e., it splits up H_2O_2 with the liberation of oxygen which oxidises guaiaconic acid into guaiacum blue. The adult's testicle has the same characters as that at the age of 15 years. The tubules ($\frac{1}{10}$ mm. in diameter) when uncoiled, are about 800 ft. long, and the spermatozoa, which are no more than $\frac{1}{500}$ in. long, are so thin that hundreds of them could easily pass side by side through the eye of a fine needle. During sexual congress they are discharged into the vagina, whence, partly by their own swimming movements at the rate of about $\frac{1}{8}$ inch per minute and possibly also by the suction action of the uterus, they travel up into the uterus and reach the Fallopian tube, where they lie in wait for the ovum to be fertilised as soon as it arrives—by the first spermatozoön that reaches it. Mott found that spermatogenesis is arrested in chronic cases of heart disease or of pulmonary tuberculosis, as well as in cases of congenital syphilis, and Kyrle found that in children who are physically undeveloped the seminiferous tubes are also undeveloped. These observations show that not only does physical development depend on testicular development, but that the arrested spermatogenesis in these cases may be a provision of nature to prevent the reproduction of the physically unfit.

In twenty-two cases of dementia præcox Mott found testicular atrophy, varying in degree from the earliest changes in the biochemical reaction of the heads of the spermatozoa to complete atrophy of the seminiferous tubules.

Relation between the Internal and External Secretions of the Testicle. —The two secretions are more or less independent of each other. In cryptorchids (undescended testicles) and in cases of ligature of the vas deferens spermatogenesis stops, but the internal secretion continues to be manufactured. The same is the case in people continually exposed (unprotected) to X-rays. Indeed, complete suppression of spermatogenesis by bilateral ligature of the vasa efferentia actually increases the internal testicular secretion and increases virility. Such operations are sometimes done for rejuvenation purposes (Steinach). Voronoff's method of rejuvenation consists in transplanting the interstitial cells of monkeys' testicles into the patient's testicles.

The reason for non-spermatogenesis, and therefore sterility, in the case of cryptorchids, seems to be the higher intra-abdominal temperature which kills the spermatozoa. It has been shown[1] that in rats the difference between the intra-abdominal and intrascrotal temperatures is as high as 7° C. From this we learn that when the testicles are incompletely descended, and are situated inside the inguinal canals, the correct treatment is to bring them down into the scrotum instead of embedding them inside the abdomen.

[1] Carl R. Moore and William J. Quick, *Amer. Journ. Phys.*, lxvii, 1924, p. 70.

The Influence of Chemical Agents upon Spermatozoa.—We saw, on p. 679, that spermatozoa are of two types—the male-producing and the female-producing. We have also seen that these two types are differently susceptible to the effects of chemical agents, since treatment of male animals with alcohol before mating kills or weakens some of the female-producing variety, and thus raises the sex ratio at birth. Yohimbine (an aphrodisiac drug) has been found to produce similar results, but the ingenious experiments of Cole and Davis [1] show that not only is there a competition in the race for fertilising the ovum between the different spermatozoa, so that the more rapid and more vigorous defeat the slower and feebler, but that chemical agents have the effect of weakening spermatozoa that are otherwise strong. These workers subjected albino female rabbits to double matings, in immediate succession as follows:—

A. (1) Coloured males (of pure coloured stock); (2) albino males (non-alcoholic). There resulted 188 offspring, of which 164 were coloured, and therefore obviously came from the coloured male (see p. 217), and 24 albinos, which equally obviously were fathered by the albinos.

B. (1) Coloured males (of pure coloured stock); (2) albino males (alcoholic). All the offspring were black, showing that the alcohol handicapped the spermatozoa of the albinos to such an extent as to render them impotent in their competition with the non-alcoholised spermatozoa of the coloured animals.

C. Cole and Davis then removed the coloured males and subjected the female albinos to matings with the alcoholised albino males alone, which then became fathers again, showing that when not competing with the non-alcoholised variety, alcoholised spermatozoa are still capable of fertilising an ovum.

(For the influence of syphilis upon the spermatozoa, see p. 111.)

Spermatoxins. Recent work[2] has shown that when animals are injected with spermatozoa, they become temporarily sterile, probably because of the production in them of an antibody, called spermatoxin, which is germicidal to spermatozoa. It is possible that quite apart from the frequency of gonorrhœa in prostitutes, which produces occlusion of the Fallopian tubes, their sterility may also be due to frequent coitus, with excessive absorption of spermatozoa through abrasions in the vaginal walls. It is equally

[1] L. J. Cole and C. L. Davis, *Science R.S.*, xxxix, 1914, p. 476.

[2] See W. Kennedy, *Quarterly Journ. Exp. Phys.*, xiv, 1924, p. 279, and J. L. McCartney, *Amer. Journ. Phys.*, lxiii, 1923, p. 237, and *ibid.*, lxvi, 1923, p. 404.

probable that very prolonged sexual abstinence might render a man temporarily or permanently sterile as the result of absorption of his own spermatozoa with the production of spermatoxin.

The mind at puberty undergoes alteration in three directions. (*a*) The will becomes temporarily more or less unbalanced, resulting in hysterical or psychical manifestations. (*b*) The intellect broadens. (*c*) The emotions become temporarily more unstable.

Epilepsy (the idiopathic variety) may manifest itself for the first time at that age, and in girls the attacks may come during a menstrual period. Heredity, however, plays a strong part in this disorder, and the disease rarely, if ever, comes in children of strong, nervous stock.

Dementia præcox, or adolescent insanity, is another condition which has its beginning at or about the period of puberty. The cause is unknown but it is believed by Mott[1] to be some toxic condition of the neurons, resulting from a deficiency of oxidation due to a want of development in the sexual glands, either directly or indirectly, through the disturbed balance of the other endocrine glands. The cause of this mental disorder, which is progressive and incurable, is unknown, but it does not seem to be of an hereditary nature and is definitely not due to syphilis, congenital or acquired.

Differences between Boys and Girls. In addition to the obvious anatomical (external and internal) and physiological differences, which are primarily associated with the function of reproduction, the sexes differ in the following respects.

(1) *Metabolic.*—The metabolic rate is on the average somewhat lower in the female than in the male (see Tables, p. 357). A high metabolic rate implies a great expenditure of energy; a low rate means a great storage of energy. Hence man uses up more and woman stores more energy out of the food they take. From this it would seem that man is intended by nature to be more actively occupied than woman, and for this reason the two sexes are not altogether suitable for exactly the same kind of education or occupation.

(2) *Endocrine.*—The internal secretion of the ovary produces the secondary female characters; that of the testes the secondary male characters, including the break in the voice (see pp. 678 and 679).

(3) *Intellectual.*—The female brain ceases to grow at about the age of 18, whilst that of the male continues growing for another ten years (see Table CCXIV, p. 630). Crichton-Browne[2] quotes Sydney Martin as stating that the internal carotid arteries which supply blood to the frontal and parietal lobes, i.e., the parts of the brain which have to do with judgment, volition and intellectual work have, on the average, a somewhat larger calibre and therefore carry more blood and oxygen in man than in woman, whilst

[1] *Loc cit.* on p. 680.

[2] "Measurement in Child Study," Presidential Address to the Conference of the Child Study Society, London, 1912.

the vertebral and basilar arteries which supply the temporo-sphenoidal and occipital lobes, i.e., the portion of the brain concerned with the reception of auditory and visual sensations, are larger in woman than in man. This agrees with Gundobin's observations that at all ages the frontal lobes are bigger in males, and the occipital lobes are bigger in females.[1] Hence it is suggested that although some women possess the highest grade of brain power, these are the exceptions, but that on the average woman cannot aspire to reach the same intellectual level as man. Whether such a contention is true or not, it is by no means easy to decide until woman has had absolutely the same opportunities for higher education, and for administrative work, as man.

(4) *Emotional.*—Inasmuch as emotions depend upon the internal secretions, we would expect to find emotional differences in the two sexes, but there is no incontrovertible evidence that such differences exist, apart from those due to differences of environment under which boys and girls are brought up.

(5) *Hæmatological.*—The number of red blood-corpuscles per cubic millimetre of blood is less in adult woman than in adult man, being on the average about $4\frac{1}{2}$ millions in the former as against 5 millions in the latter. In other words, per unit volume of blood, woman is 10 per cent poorer in the red corpuscles, and therefore in oxygen-carrying hæmoglobin, than man. This difference, however, does not apply to infancy and school age. During infancy the number seems to be the same in both sexes, whilst in children at the age of 12 there is apparently a greater number of red cells in girls than in boys. This may be accounted for by the greater rate of growth of girls at that age (see fig. 149), and unless special care is taken with regard to their physical and mental occupations during the pubescent period, they are very liable to become markedly anæmic.

(6) *Cellular.*—Recent work tends to show that all the above and other peculiarities which differentiate men from women are ultimately associated with the difference in the chromosome constitution of the cells in man and in woman. We have seen that the soma cell of the female contains two x-chromosomes, whilst that of the male contains one x and one y-chromosome. The y-chromosome is associated with a higher metabolic rate. It also seems to carry with it some lethal factor which renders the death-rate amongst males at all ages—including the fœtal and embryonic stage—higher than amongst females, and thus makes the expectation of life at all ages higher in females than in males (see pp. 179 and 180). But for this higher ante-natal mortality amongst males, the sex ratio at birth would, as we have seen, be considerably higher than it is.

Hygiene of Adolescence.—Apart from the extra amount of sleep and food required during this period, there is nothing special to be said about the hygiene of puberty. The awakening of the sexual instinct, however, is a phenomenon on which the child needs a few words of explanation and preparation. The girl should be prepared by her mother about the onset of menstruation, (since to the uninstructed girl the first menstrual period may come

[1] N. P. Gundobin, "Die Besonderheiten des Kindesalters," Berlin, 1912, p. 468.

as a shock), and at the same time a tactful and cultured mother might take an opportunity of explaining to her daughter the significance of the phenomenon as a physiological preparation for motherhood, that when impregnation occurs menstruation ceases, but that it may also cease in cases of ill-health apart from pregnancy. The girl should also be informed that the condition is not a pathological phenomenon, but is an absolutely normal and natural occurrence, and that in the majority of cases there is no reason why the usual duties, including *moderate* exercise, should not be performed during the menstrual period. Boys should be told by their fathers that it is possible that they might get an involuntary emission of semen during sleep (wet dream), and that this is a phenomenon which should not alarm them, as it is a perfectly natural and physiological occurrence.

The Biblical injunction regarding the semi-isolation of a menstruating woman has recently been explained on a scientific basis. It has been found that during her menstrual period, her blood, saliva, perspiration and milk contain a toxic material which is harmful to living tissue, especially plants, so that a flower held by a menstruating woman will wilt more quickly than one held at any other time. The toxin in the milk may also explain the occasional disturbance of the nursling during the mother's menstrual period.[1]

Sex Education.—Both boys and girls should receive instruction from their parents on the phenomena of sex and reproduction, and given a few words of advice with regard to the necessity of self-control in all matters, especially in those relating to the gratification of the sexual passion. It must be pointed out to them that they must discipline themselves in such a way as to be the masters and not the slaves of any impulse. It should be stated quite frankly that there is nothing wrong in sexual gratification as such, but that the evil lies in its premature or illicit gratification. The premature gratification of the act is injurious because the genital organs are not yet mature, and perfect function of any organ is incompatible with its incomplete maturity. It is, therefore, as bad as feeding an infant on food that it is, as yet, unable to digest. The same remarks apply to masturbation, which is not only harmful in that its excessive practice may bring about a weakening of the organ so that it is unable to function normally later on, but that it is a sign of impaired self-discipline. We have seen (p. 681) that when spermatogenesis

[1] See David I. Macht and Dorothy G. Lubin, *Journ. Pharm. and Exper. Therap.*, 1924, xxii, p. 413.

ceases, the internal secretion of the testicle increases. Hence it is reasonable to believe that when spermatogenesis is increased the testicular hormone may become diminished, and if this happens before complete maturity both physical and mental growth may suffer. Boys especially must be informed that illicit intercourse, apart from weakening the moral and disciplinary character, is attended by grave risks of venereal diseases and their consequences, not only to themselves but to their future wives and families.

The physical and spiritual evils of associating with loose women are most cogently put by King Solomon (Prov. ch. vii) and might be profitably committed to memory by every child of school-leaving age.

"My son keep my words and lay up my commandments with thee. . . . Write them upon the table of thine heart . . . that they may keep thee from the strange woman. . . . Let not thine heart decline to her ways. For she hath cast down many wounded, yea many strong men have been slain by her. Her house is the way to hell going down to the chamber of death."

The dangers of venereal disease should be explained, but an appeal to the boy's sense of honour is probably more effective. The dangers of alcohol in stimulating sexual passions and in reducing the inhibitory control of the brain should also be explained, and the boy should, therefore, be encouraged in every possible way to eschew spices and all kinds of alcoholic drinks. Pornographic literature, even when disguised under classical or scientific titles, should be forbidden.

With children whose physical and mental upbringing has been along the right lines, as laid down in the various chapters of this book, there should be no great difficulty on the score of sexual enlightenment or frank speaking at this critical age. Let the young adolescent ever bear in mind the words of Ecclesiastes (xi. 9).

"Rejoice, O young man, in thy youth, and let thy heart cheer thee in the days of thy youth, and walk in the ways of thine heart, and in the sight of thine eyes; but know thou that for all these things God will bring thee into judgment."

We have now completed the cycle of child life. We started from the ante-conceptional stage, when the child still had a dual existence inside the germ cells of its parents, and have passed in review every phase from that stage onwards until the stage of puberty, which is the ante-conceptional stage of the next generation. The genetic relationship between parent and child is

poetically expressed in the following lines, by Dr. T. Lovell Beddoes, and with these we may fittingly close the book :—

> "There stands before you
> The youth and golden top of your existence,
> Another life of yours : for think your morning
> Not lost but given, pass'd from your hand to his,
> The same except in place. Be then to him
> As the former tenant of your age,
> When you were in the prologue of your time
> And he lay hid unconsciously
> Under his life. And thou, my young master,
> Remember there's a kind of God in him ;
> And after heaven, the next of thy religion,
> Thy second fear of God, thy first of man
> Are his, who was creation's delegate
> And made this world for thee, in making thee."

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